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SEARCH REQUEST FORM

Requestor's Name: Geetha Bansal

Serial Number: ~~09/~~ 09/522,716

Date: 4/17/01 Phone: 8E12 Art Unit: 1642

Search Topic:

Please write a detailed statement of search topic. Describe specifically as possible the subject matter to be searched. Define any terms that may have a special meaning. Give examples or relevent citations, authors, keywords, etc., if known. For sequences, please attach a copy of the sequence. You may include a copy of the broadest and/or most relevent claim(s).

Please search:

1. a semi allogeneic cell, hybrid formed by fusing an Antigen presenting cell (APC) with a tumor cell. and the hybrid cell expresses class I or II allogeneic to the recipient and one class I or II syngeneic to recipient

2. Use of above cell in treating or preventing cancer

RECEIVED MAY 22 2001 (STIC)

Thanks

POINT OF CONTACT:
BARB O'BRYEN
TECH. INFORMATION SPECIALIST
STIC CM1 12C14 308-4291

Cancer Immunotherapy w/ semi-allogeneic cells
Cohen, E.

STAFF USE ONLY

Date completed: 6-26-01
Searcher: BOBB
Terminal time: 73
Elapsed time: prep 25
CPU time:
Total time:
Number of Searches:
Number of Databases:

Search Site
- ___ STIC
- ___ CM-1
- ___ Pre-S

Type of Search
- ___ N.A. Sequence
- ___ A.A. Sequence
- ___ Structure
- X Bibliographic

Vendors
- ___ IG
- ___ STN
- ___ Dialog
- ___ APS
- ___ Geninfo
- ___ SDC
- ___ DARC/Questel
- ___ Other

PTO-1590 (9-90)

=> fil capl; d que l15; d que l20; d que l28; s l15 or l20 or l28; fil cancer
FILE 'CAPLUS' ENTERED AT 12:00:12 ON 26 JUN 2001

FILE COVERS 1947 - 26 Jun 2001 VOL 135 ISS 1
FILE LAST UPDATED: 25 Jun 2001 (20010625/ED)

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This file supports REG1stRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

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The CA Lexicon is now available in the Controlled Term (/CT) field. Enter HELP LEXICON for full details.

L2	7434	SEA	FILE=CAPLUS ABB=ON	ALLOGENEIC
L4	56	SEA	FILE=CAPLUS ABB=ON	SEMI(W)L2
L7	4061	SEA	FILE=CAPLUS ABB=ON	IMMUNOTHERAPY+OLD/CT
L14	115	SEA	FILE=CAPLUS ABB=ON	SEMIALLOGEN?
L15	5	SEA	FILE=CAPLUS ABB=ON	(L14 OR L4) AND L7

L2	7434	SEA	FILE=CAPLUS ABB=ON	ALLOGENEIC
L4	56	SEA	FILE=CAPLUS ABB=ON	SEMI(W)L2
L14	115	SEA	FILE=CAPLUS ABB=ON	SEMIALLOGEN?
L20	3	SEA	FILE=CAPLUS ABB=ON	(L4 OR L14)(L)THU/RL

-Role - therapeutic use

L6	11208	SEA	FILE=CAPLUS ABB=ON	ANTIGEN PRESENT?
L8	20050	SEA	FILE=CAPLUS ABB=ON	TUMORS/CW
L9	38898	SEA	FILE=CAPLUS ABB=ON	HLA OR MHC
L10	21474	SEA	FILE=CAPLUS ABB=ON	HISTOCOMPATIBILITY/OBI
L11	141791	SEA	FILE=CAPLUS ABB=ON	(FUSION OR FUSED OR FUSING)/OBI
L12	61907	SEA	FILE=CAPLUS ABB=ON	TUMOR(A)CELL#
L13	12125	SEA	FILE=CAPLUS ABB=ON	?ALLOGENEIC? OR ?ALLOGENIC?
L16	209372	SEA	FILE=CAPLUS ABB=ON	HYBRID?
L18	906	SEA	FILE=CAPLUS ABB=ON	TUMOUR#

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L22      187332 SEA FILE=CAPLUS ABB=ON  NEOPLASM/CW
L23      114744 SEA FILE=CAPLUS ABB=ON  ANTITUMOR AGENTS+OLD/CT
L27       40399 SEA FILE=CAPLUS ABB=ON  DENDRITIC OR LANGERHANS
L28           7 SEA FILE=CAPLUS ABB=ON  (L6 OR L27) AND L13 AND (L9 OR L10)
                AND (L16 OR L11) AND (L8 OR L12 OR L18 OR L22 OR L23)


L129         13 L15 OR L20 OR L28


FILE 'CANCERLIT' ENTERED AT 12:00:14 ON 26 JUN 2001

 FILE COVERS 1963 TO 14 Jun 2001 (20010614/ED)

 CANCERLIT thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2000 vocabulary.  Enter HELP THESAURUS for details.

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 The CANCERLIT citations and abstracts for December, January, February, and
 March are not yet available due to a delay in receiving the source data
 from the National Cancer Institute.  Once received and processed, all
 monthly updates for CANCERLIT will be provided.

=> d que l36; d que l47; d que l51; d que l52; d que l59; d que l64
L25          194 SEA FILE=CANCERLIT ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC?
                 OR ALLOGENIC?)
L31         1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32         6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L34       134884 SEA FILE=CANCERLIT ABB=ON  TUMOR CELLS, CULTURED+NT/CT
L36            3 SEA FILE=CANCERLIT ABB=ON  L25 AND (L31 OR L32) AND L34


L31         1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32         6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L38        14727 SEA FILE=CANCERLIT ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L46         1363 SEA FILE=CANCERLIT ABB=ON  CANCER VACCINES/CT
L47            7 SEA FILE=CANCERLIT ABB=ON  L38 AND (L31 OR L32) AND L46


L26         5914 SEA FILE=CANCERLIT ABB=ON  ANTIGEN-PRESENTING CELLS+NT/CT
L31         1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32         6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L34       134884 SEA FILE=CANCERLIT ABB=ON  TUMOR CELLS, CULTURED+NT/CT
L38        14727 SEA FILE=CANCERLIT ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L44         1565 SEA FILE=CANCERLIT ABB=ON  ANTIGEN PRESENTATION/CT
L51            2 SEA FILE=CANCERLIT ABB=ON  (L26 OR L44) AND L38 AND (L31 OR
                 L32) AND L34


L25          194 SEA FILE=CANCERLIT ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC?
                 OR ALLOGENIC?)
L31         1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32         6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L35           10 SEA FILE=CANCERLIT ABB=ON  L25 AND (L31 OR L32)
L52            2 SEA FILE=CANCERLIT ABB=ON  L35 AND FIBROSARCOMA/CT
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          194 SEA FILE=CANCERLIT ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC?
              OR ALLOGENIC?)
L29         16934 SEA FILE=CANCERLIT ABB=ON  HISTOCOMPATIBILITY ANTIGENS+NT/CT
L30          3364 SEA FILE=CANCERLIT ABB=ON  MAJOR HISTOCOMPATIBILITY COMPLEX+NT/
              CT
L31          1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32          6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L37         17255 SEA FILE=CANCERLIT ABB=ON  ANTIGENS, NEOPLASM/CT
L59             2 SEA FILE=CANCERLIT ABB=ON  L25 AND (L29 OR L30) AND (L31 OR
              L32) AND L37



L26          5914 SEA FILE=CANCERLIT ABB=ON  ANTIGEN-PRESENTING CELLS+NT/CT
L29         16934 SEA FILE=CANCERLIT ABB=ON  HISTOCOMPATIBILITY ANTIGENS+NT/CT
L30          3364 SEA FILE=CANCERLIT ABB=ON  MAJOR HISTOCOMPATIBILITY COMPLEX+NT/
              CT
L31          1613 SEA FILE=CANCERLIT ABB=ON  CELL FUSION/CT
L32          6796 SEA FILE=CANCERLIT ABB=ON  HYBRID CELLS+NT/CT
L33         22876 SEA FILE=CANCERLIT ABB=ON  IMMUNOTHERAPY+NT/CT
L34        134884 SEA FILE=CANCERLIT ABB=ON  TUMOR CELLS, CULTURED+NT/CT
L37         17255 SEA FILE=CANCERLIT ABB=ON  ANTIGENS, NEOPLASM/CT
L44          1565 SEA FILE=CANCERLIT ABB=ON  ANTIGEN PRESENTATION/CT
L63         55717 SEA FILE=CANCERLIT ABB=ON  TRANSPLANTATION+NT/CT
L64             5 SEA FILE=CANCERLIT ABB=ON  (L26 OR L44 OR L37) AND L33 AND
              (L29 OR L30) AND (L31 OR L32) AND (L34 OR L63)


=> s l36 or l47 or l51 or l52 or l59 or l64
L130          18 L36 OR L47 OR L51 OR L52 OR L59 OR L64

=> fil wpids; d que l67; d que l74; d que l79; s l67 or l74 or l79; fil biosis
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FILE LAST UPDATED: 25 JUN 2001              <20010625/UP>
MOST RECENT DERWENT UPDATE            200135   <200135/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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L65             6 SEA FILE=WPIDS ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC? OR
              ALLOGENIC?)
L66        118142 SEA FILE=WPIDS ABB=ON  HYBRID? OR FUSION OR FUSED OR FUSING
L67             3 SEA FILE=WPIDS ABB=ON  L65 AND L66
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L66      118142 SEA FILE=WPIDS ABB=ON  HYBRID? OR FUSION OR FUSED OR FUSING
L68         497 SEA FILE=WPIDS ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L69        1598 SEA FILE=WPIDS ABB=ON  HLA OR MHC OR HISTOCOMPATIBILITY
L70         675 SEA FILE=WPIDS ABB=ON  ANTIGEN PRESENT?
L72        1644 SEA FILE=WPIDS ABB=ON  DENDRITIC OR LANGERHANS
L74           4 SEA FILE=WPIDS ABB=ON  L66 AND L68 AND L69 AND (L70 OR L72)


L66      118142 SEA FILE=WPIDS ABB=ON  HYBRID? OR FUSION OR FUSED OR FUSING
L68         497 SEA FILE=WPIDS ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L71          55 SEA FILE=WPIDS ABB=ON  L66 AND L68
L75        1049 SEA FILE=WPIDS ABB=ON  IMMUNOTHERAP? OR IMMUNO THERAP?
L77       47096 SEA FILE=WPIDS ABB=ON  CANCER? OR TUMOR# OR TUMOUR#
L79           4 SEA FILE=WPIDS ABB=ON  L71 AND L77 AND L75


L131          9 L67 OR L74 OR L79


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CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 20 June 2001 (20010620/ED)

The BIOSIS file has been reloaded.  Enter HELP RLOAD and HELP REINDEXING
for details.

=> d que l90; d que l94; d que l101; d que l107; s l90 or l94 or l101 or l107
L83          498 SEA FILE=BIOSIS ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC? OR
                 ALLOGENIC?)
L85        23510 SEA FILE=BIOSIS ABB=ON  IMMUNOTHERAP?
L87       287059 SEA FILE=BIOSIS ABB=ON  FUSION OR FUSED OR FUSING OR HYBRID?
L90            4 SEA FILE=BIOSIS ABB=ON  L83 AND L87 AND L85


L83          498 SEA FILE=BIOSIS ABB=ON  SEMIALLOGEN? OR SEMI(W)(ALLOGENEIC? OR
                 ALLOGENIC?)
L85        23510 SEA FILE=BIOSIS ABB=ON  IMMUNOTHERAP?
L87       287059 SEA FILE=BIOSIS ABB=ON  FUSION OR FUSED OR FUSING OR HYBRID?
L91       936854 SEA FILE=BIOSIS ABB=ON  CANCER? OR NEOPLAS? OR TUMOR? OR
                 TUMOUR?
L94            4 SEA FILE=BIOSIS ABB=ON  L83 AND L85 AND L87 AND L91


L84        43490 SEA FILE=BIOSIS ABB=ON  ANTIGEN PRESENT? OR DENDRITIC OR
                 LANGERHANS
L85        23510 SEA FILE=BIOSIS ABB=ON  IMMUNOTHERAP?
L86        94499 SEA FILE=BIOSIS ABB=ON  HLA OR MHC OR HISTOCOMPATIBILITY
L87       287059 SEA FILE=BIOSIS ABB=ON  FUSION OR FUSED OR FUSING OR HYBRID?
L91       936854 SEA FILE=BIOSIS ABB=ON  CANCER? OR NEOPLAS? OR TUMOR? OR
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              TUMOUR?
          26794 SEA FILE=BIOSIS ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
             44 SEA FILE=BIOSIS ABB=ON  L95 AND L84 AND L86 AND L87
              3 SEA FILE=BIOSIS ABB=ON  L97 AND L91 AND L85


          43490 SEA FILE=BIOSIS ABB=ON  ANTIGEN PRESENT? OR DENDRITIC OR
              LANGERHANS
L86       94499 SEA FILE=BIOSIS ABB=ON  HLA OR MHC OR HISTOCOMPATIBILITY
L87      287059 SEA FILE=BIOSIS ABB=ON  FUSION OR FUSED OR FUSING OR HYBRID?
L91      936854 SEA FILE=BIOSIS ABB=ON  CANCER? OR NEOPLAS? OR TUMOR? OR
              TUMOUR?
L95       26794 SEA FILE=BIOSIS ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L97          44 SEA FILE=BIOSIS ABB=ON  L95 AND L84 AND L86 AND L87
L102     911367 SEA FILE=BIOSIS ABB=ON  ?THERAP?
L106      33160 SEA FILE=BIOSIS ABB=ON  (IMMUNE(W)(RESPONSE OR SPECIFIC))/IT
L107          3 SEA FILE=BIOSIS ABB=ON  L97 AND L91 AND L102 AND L106


L132          9 L90 OR L94 OR L101 OR L107

=> fil embase; d que l119; d que l123; d que l127; s l119 or l123 or l127
FILE 'EMBASE' ENTERED AT 12:01:50 ON 26 JUN 2001
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 FILE COVERS 1974 TO 22 Jun 2001 (20010622/ED)

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L109       8084 SEA FILE=EMBASE ABB=ON  ANTIGEN PRESENTATION/CT
L110     103166 SEA FILE=EMBASE ABB=ON  ANTIGEN PRESENTING CELL+NT/CT
L111       2408 SEA FILE=EMBASE ABB=ON  HYBRID/CT
L112       7085 SEA FILE=EMBASE ABB=ON  HYBRID CELL/CT
L113       3928 SEA FILE=EMBASE ABB=ON  CELL FUSION/CT
L114      47523 SEA FILE=EMBASE ABB=ON  HISTOCOMPATIBILITY ANTIGEN+NT/CT
L115      45873 SEA FILE=EMBASE ABB=ON  MAJOR HISTOCOMPATIBILITY ANTIGEN+NT/CT

L117      24656 SEA FILE=EMBASE ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L118         91 SEA FILE=EMBASE ABB=ON  (L109 OR L110) AND ((L111 OR L112 OR
              L113)) AND (L114 OR L115)
L119          9 SEA FILE=EMBASE ABB=ON  L118 AND L117


L111       2408 SEA FILE=EMBASE ABB=ON  HYBRID/CT
L112       7085 SEA FILE=EMBASE ABB=ON  HYBRID CELL/CT
L113       3928 SEA FILE=EMBASE ABB=ON  CELL FUSION/CT
L117      24656 SEA FILE=EMBASE ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L121      16520 SEA FILE=EMBASE ABB=ON  IMMUNOTHERAPY/CT
L123          1 SEA FILE=EMBASE ABB=ON  ((L111 OR L112 OR L113)) AND L121 AND
              L117


L111       2408 SEA FILE=EMBASE ABB=ON  HYBRID/CT
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L112        7085 SEA FILE=EMBASE ABB=ON  HYBRID CELL/CT
L113        3928 SEA FILE=EMBASE ABB=ON  CELL FUSION/CT
L117       24656 SEA FILE=EMBASE ABB=ON  ?ALLOGENEIC? OR ?ALLOGENIC?
L125       14763 SEA FILE=EMBASE ABB=ON  CANCER IMMUNIZATION/CT OR CANCER
                 IMMUNOTHERAPY/CT OR CANCER VACCINE/CT
L127           7 SEA FILE=EMBASE ABB=ON  ((L111 OR L112 OR L113)) AND L117 AND
                 L125


L133          16 L119 OR L123 OR L127

=> dup rem l130,l129,l132,l133,l131
FILE 'CANCERLIT' ENTERED AT 12:02:08 ON 26 JUN 2001

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PROCESSING COMPLETED FOR L130
PROCESSING COMPLETED FOR L129
PROCESSING COMPLETED FOR L132
PROCESSING COMPLETED FOR L133
PROCESSING COMPLETED FOR L131
L134           46 DUP REM L130 L129 L132 L133 L131 (19 DUPLICATES REMOVED)
                  ANSWERS '1-18' FROM FILE CANCERLIT
                  ANSWERS '19-26' FROM FILE CAPLUS
                  ANSWERS '27-31' FROM FILE BIOSIS
                  ANSWERS '32-40' FROM FILE EMBASE
                  ANSWERS '41-46' FROM FILE WPIDS

=> d ibib ab 1-46; fil hom

L134 ANSWER 1 OF 46  CANCERLIT                          DUPLICATE 2
ACCESSION NUMBER:    2001078527  CANCERLIT
DOCUMENT NUMBER:     21078527
TITLE:               Semiallogeneic cancer vaccines formulated with
                     granulocyte-macrophage colony-stimulating factor for
                     patients with metastatic gastrointestinal adenocarcinomas:
                     a pilot phase I study.
AUTHOR:              Newton D A; Acierno P M; Metts M C; Baron P L; Brescia F J;
                     Gattoni-Celli S
CORPORATE SOURCE:    Department of Radiation Oncology, Hollings Cancer Center,
                     Medical University of South Carolina, Charleston, USA.
SOURCE:              JOURNAL OF IMMUNOTHERAPY, (2001). Vol. 24, No. 1, pp.
                     19-26.
                     Journal code: CUQ.
DOCUMENT TYPE:       (CLINICAL TRIAL)
                     (CLINICAL TRIAL, PHASE I)
                     Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT:        MEDL; L; I
LANGUAGE:            English
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SOURCE: MEDLINE 21078527
MONTH: 200104
The authors report the results of a phase I clinical study using **semiallogeneic** cancer vaccines formulated with granulocyte-macrophage colony-stimulating factor (GM-CSF) to treat patients with metastatic adenocarcinomas of the gastrointestinal tract. A specially engineered cell line, FO1-12, was used to generate **semiallogeneic** hybrids by fusion with patient-derived tumor cells; the hybrids express HLA class I and II haplotypes derived from both parental cells. For treatment, the vaccine was mixed with GM-CSF, irradiated, and injected intradermally into patients at weekly or biweekly intervals. Vaccinations were associated with minimal or no toxicity and showed that **semiallogeneic** hybrids formulated with GM-CSF can induce a specific antitumor immune response in some patients, as measured by a delayed-type hypersensitivity response to autologous tumor cells. Because of the simplicity, feasibility, and flexibility of this immunotherapeutic approach, **semiallogeneic** hybrid vaccines have the potential to be used in the treatment of virtually any type of cancer.

L134 ANSWER 2 OF 46 CANCERLIT DUPLICATE 4
ACCESSION NUMBER: 2000436913 CANCERLIT
DOCUMENT NUMBER: 20436913
TITLE: **Semi-allogeneic** cell hybrids stimulate HIV-1 envelope-specific cytotoxic T lymphocytes.
AUTHOR: Grene E; Newton D A; Brown E A; Berzofsky J A; Gattoni-Celli S; Shearer G M
CORPORATE SOURCE: Experimental Immunology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892, USA.
SOURCE: AIDS, (2000). Vol. 14, No. 11, pp. 1497-506. Journal code: AID. ISSN: 0269-9370.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; I
LANGUAGE: English
OTHER SOURCE: MEDLINE 20436913
ENTRY MONTH: 200103
AB OBJECTIVE: The present study was designed to determine whether the HLA allogeneic T helper response stimulated by **semi-allogeneic** cell lines could be used as an in vitro model of immune-based therapy to stimulate HIV-specific cytotoxic T lymphocytes. DESIGN AND METHODS: **Semi-allogeneic** cell hybrids were obtained by the fusion of peripheral blood mononuclear cells from HIV-infected patients with the allogeneic beta2-microglobulin-deficient FO1-12 melanoma cell line. These hybrids were used as antigen presenting cells for HIV envelope peptide (env)-specific cytotoxic assays. RESULTS: The hybrid cell lines express HLA class I and II antigens from both parental cells, as well as the CD86 costimulatory molecule. HIV-specific cytotoxic T lymphocyte activity was obtained when patients' peripheral blood mononuclear cells were costimulated with env peptides plus **semi-allogeneic** hybrids, in contrast with stimulation with either env or hybrid cells alone. Thus, the **semi-allogeneic** hybrids enhanced HIV-specific killing of target cells. CONCLUSIONS: Irradiated, **semi-allogeneic** cell hybrids engineered for individual AIDS patients provide efficient and simultaneous co-recognition of HLA allogeneic determinants and viral antigenic determinants presented by self-HLA molecules on the same antigen presenting cells and results in the generation of enhanced HIV-specific cytotoxic T lymphocyte activity.

L134 ANSWER 3 OF 46 CANCERLIT DUPLICATE 5
ACCESSION NUMBER: 2000164583 CANCERLIT

DOCUMENT NUMBER: 20164583
TITLE: Hybrid cell vaccination for cancer immune therapy: first clinical trial with metastatic melanoma.
AUTHOR: Trefzer U; Weingart G; Chen Y; Herberth G; Adrian K; Winter H; Audring H; Guo Y; Sterry W; Walden P
CORPORATE SOURCE: Department of Dermatology, Medical Faculty Charite, Humboldt University, Berlin, Germany.
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (2000). Vol. 85, No. 5, pp. 618-26.
Journal code: GQU. ISSN: 0020-7136.
DOCUMENT TYPE: (CLINICAL TRIAL)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL, PHASE II)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 20164583
ENTRY MONTH: 200004
AB Hybrid cell vaccination is a new cancer immune therapy approach that aims at recruiting T cell help for the induction of tumour specific cytolytic immunity. The vaccines are generated by fusion of the patients' tumour cells with **allogeneic** MHC class II bearing cells to combine the tumour's antigenicity with the immunogenicity of **allogeneic** MHC molecules. Safety and anti-tumour activity of this treatment were assessed in a clinical trial that has yielded one complete and one partial remission, and 5 cases of stable disease among 16 patients with advanced stage metastatic melanoma. As evidenced by histology, the vaccination induced T cell relocation into tumour nodules. Stable disease could be maintained by repeated booster injections for more than 24 months in some patients. The side effects were minor. Occasional occurrences of vitiligo spots after vaccination were indicative of a restricted therapy induced auto-immune reactivity. The results suggest that hybrid cell vaccination is a safe cancer immune therapy potentially effective for induction of acute anti-tumour response as well as long-term maintenance. Copyright 2000 Wiley-Liss, Inc.

L134 ANSWER 4 OF 46 CANCERLIT DUPLICATE 6
ACCESSION NUMBER: 2000165230 CANCERLIT
DOCUMENT NUMBER: 20165230
TITLE: Regression of human metastatic renal cell carcinoma after vaccination with tumor cell-dendritic cell hybrids [see comments].
COMMENT: Comment in: Nat Med 2000 Mar;6(3):252-3
AUTHOR: Kugler A; Stuhler G; Walden P; Zoller G; Zobywalski A; Brossart P; Trefzer U; Ullrich S; Muller C A; Becker V; Gross A J; Hemmerlein B; Kanz L; Muller G A; Ringert R H
CORPORATE SOURCE: Department of Urology, University of Gottingen, Germany. akugler@gwdg.de
SOURCE: NATURE MEDICINE, (2000). Vol. 6, No. 3, pp. 332-6.
Journal code: CG5. ISSN: 1078-8956.
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 20165230
ENTRY MONTH: 200004
AB Reports of spontaneous regressions of metastases and the demonstration of tumor-reactive cytotoxic T lymphocytes indicate the importance of the host's immune system in controlling the devastating course of metastatic renal cell carcinoma. Recent research indicates that immunization with

ybrids of tumor and antigen presenting cells results in protective immunity and rejection of established tumors in various rodent models. Here, we present a hybrid cell vaccination study of 17 patients. Using electrofusion techniques, we generated hybrids of autologous tumor and **allogeneic** dendritic cells that presented antigens expressed by the tumor in concert with the co-stimulating capabilities of dendritic cells. After vaccination, and with a mean follow-up time of 13 months, four patients completely rejected all metastatic tumor lesions, one presented a 'mixed response', and two had a tumor mass reduction of greater 50%. We also demonstrate induction of HLA-A2-restricted cytotoxic T cells reactive with the Muc1 tumor-associated antigen and recruitment of CD8+ lymphocytes into tumor challenge sites. Our data indicate that hybrid cell vaccination is a safe and effective therapy for renal cell carcinoma and may provide a broadly applicable strategy for other malignancies with unknown antigens.

L134 ANSWER 5 OF 46 CANCERLIT DUPLICATE 7
ACCESSION NUMBER: 2000208340 CANCERLIT
DOCUMENT NUMBER: 20208340
TITLE: **Semiallogeneic** cell hybrids as therapeutic vaccines for cancer.
AUTHOR: Newton D A; Romano C; Gattoni-Celli S
CORPORATE SOURCE: Department of Radiation Oncology, Hollings Cancer Center, Medical University of South Carolina, Charleston 29403, USA

SOURCE: JOURNAL OF IMMUNOTHERAPY, (2000). Vol. 23, No. 2, pp. 246-54.
Journal code: CUQ.
DOCUMENT TYPE: (CLINICAL TRIAL)
(CLINICAL TRIAL, PHASE I)
(CLINICAL TRIAL, PHASE II)
Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 20208340
ENTRY MONTH: 200007
AB The authors have engineered a cell line that can be used in human studies as a universal donor cell for the formation of **semiallogeneic** cell hybrids after fusion with patient-derived tumor cells. These hybrids can be irradiated and injected as a patient-tailored therapeutic vaccine in patients affected by virtually any type of cancer. A crucial step in this research effort has been the derivation of an **allogeneic** cell line (FO1-12) that expresses both a dominant selectable marker (neomycin resistance) and a recessive selectable marker (sensitivity to hypoxanthine, aminopterin, and thymidine), which allows easy selection of **semiallogeneic** cell hybrids derived from the fusion of FO1-12 cells with patient-derived tumor cells. Tumor-infiltrating lymphocytes derived from select patients with melanoma and exposed to **semiallogeneic** cell hybrids from the same patient were better able to specifically lyse autologous tumor cells. Furthermore, FO1-12 cells express carcinoembryonic antigen, which is ubiquitous in adenocarcinomas, and fusion of FO1-12 cells with various patient-derived adenocarcinoma cells showed that the hybrid cells also express carcinoembryonic antigen. Because of the results of these preclinical studies, the authors were given permission to use **semiallogeneic** cell hybrids for immunotherapy of patients with metastatic melanoma or metastatic adenocarcinoma who had not responded to standard treatment regimens. Treatment with **semiallogeneic** vaccines is associated with minimal or no toxicity and can induce a specific anti-tumor immune response.

L134 ANSWER 6 OF 46 CANCERLIT DUPLICATE 8
ACCESSION NUMBER: 2000062759 CANCERLIT
DOCUMENT NUMBER: 20062759
TITLE: Human antigen-presenting cell/tumour cell hybrids stimulate strong **allogeneic** responses and present tumour-associated antigens to cytotoxic T cells in vitro.
AUTHOR: Dunnion D J; Cywinski A L; Tucker V C; Murray A K; Rickinson A B; Coulie P; Browning M J
CORPORATE SOURCE: Department of Microbiology, Leicester University, UK.
SOURCE: IMMUNOLOGY, (1999). Vol. 98, No. 4, pp. 541-50. Journal code: GH7. ISSN: 0019-2805.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 20062759
ENTRY MONTH: 200003
AB Most tumours do not stimulate effective antitumour immune responses in vivo. In order to enhance the immunogenicity of human tumour cells, we fused a variety of tumour cell lines with an Epstein-Barr virus transformed B-lymphoblastoid cell line (EBV B-LCL) in vitro, to produce stable hybrid cells. Hybrid cell lines showed a marked increase in their ability to stimulate primary **allogeneic** T-cell responses in vitro, as compared with the parent tumour cells. The hybrid cells induced proliferation of naive (CD45RA+) as well as memory (CD45RO+) T lymphocytes, and both CD4+ and CD8+ subpopulations of T cells were directly stimulated. The stimulatory hybrids expressed human leucocyte antigen (HLA) class I and II, and a wide range of surface accessory molecules, including the T-cell co-stimulatory ligand molecules CD40, CD80 (B7.1) and CD86 (B7.2), the expression of which was required for optimal stimulation of T-cell responses. Fusion of the EBVB-LCL with a melanoma cell line (518.A2) yielded hybrid cells that expressed the melanoma-associated antigens MAGE-1 and MAGE-3, and presented these antigens to antigen-specific, HLA class I-restricted cytotoxic T-lymphocyte clones with greater efficiency than the parent melanoma cell line. These findings suggest that the generation of human antigen-presenting cell/tumour cell hybrids offers promise as an approach to cancer immunotherapy.

L134 ANSWER 7 OF 46 CANCERLIT DUPLICATE 11
ACCESSION NUMBER: 1999021012 CANCERLIT
DOCUMENT NUMBER: 99021012
TITLE: Autologous and **allogenic** hybrid cell vaccine in patients with metastatic renal cell carcinoma.
AUTHOR: Kugler A; Seseke F; Thelen P; Kallerhoff M; Muller G A; Stuhler G; Muller C; Ringert R H
CORPORATE SOURCE: Department of Urology, University of Gottingen, Germany.
SOURCE: BRITISH JOURNAL OF UROLOGY, (1998). Vol. 82, No. 4, pp. 487-93. Journal code: B3K. ISSN: 0007-1331.
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 99021012
ENTRY MONTH: 199901
AB OBJECTIVE: To evaluate the safety, acute and long-term toxicity and therapeutic activity of an **allogenic** and an autologous hybrid cell vaccine in patients with progressive metastatic renal cell carcinoma (RCC). PATIENTS AND METHODS: Eleven patients were vaccinated with a lethally irradiated hybrid cell vaccine of **allogenic** RCC tumour cells fused with major histocompatibility complex class I-matched and

class II-unmatched activated **allogenic** lymphocytes. These patients were then followed for a mean of 11 months. Another 13 patients were vaccinated with a hybrid cell vaccine of autologous tumour cells fused with **allogenic** activated lymphocytes and followed for a mean of 6 months. RESULTS: Six of the 11 patients receiving the **allogenic** vaccination showed an initial response, with two complete and two partial responses to date. Only three patients who received autologous vaccination responded to treatment. CONCLUSIONS: Hybrid cell vaccination is a promising new approach in the treatment of patients with advanced RCC.

L134 ANSWER 8 OF 46 CANCERLIT DUPLICATE 12
ACCESSION NUMBER: 1998102817 CANCERLIT
DOCUMENT NUMBER: 98102817
TITLE: Co-expression of immunogenic determinants by the same cellular immunogen is required for the optimum immunotherapeutic benefit in mice with melanoma.
AUTHOR: Xu W; de Zoeten E; Carr-Brendel V; Cohen E P
CORPORATE SOURCE: Department of Microbiology and Immunology (M/C 790), Chicago, IL 60612, USA.
SOURCE: CANCER IMMUNOLOGY, IMMUNOTHERAPY, (1998). Vol. 45, No. 5, pp. 217-24.
Journal code: CN3. ISSN: 0340-7004.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 98102817
ENTRY MONTH: 199803
AB Tumor-associated T cell epitopes are recognized by T cells in the context of determinants specified by class I loci. Since the rejection of foreign histocompatibility antigens is known to enhance tumor immunity, immunization with a cellular vaccine that combined the expression of both syngeneic and **allogeneic** class I determinants could have important immunological advantages over a vaccine that expressed either syngeneic or **allogeneic** determinants alone. To investigate this question in a mouse melanoma model system, we tested the immunotherapeutic properties of B16 melanoma x LM fibroblast hybrid cells in C57BL/6J mice with melanoma. Like C57BL/6J mice, B16 cells expressed H-2Kb class I determinants and (antibody-defined) melanoma-associated antigens. LM cells, of C3H mouse origin, formed H-2Kk determinants along with B7.1, a co-stimulatory molecule that can activate T cells. The B16 x LM hybrid cells co-expressed H-2Kb and H-2Kk class I determinants, B7.1 and the melanoma-associated antigens. C57BL/6J mice with melanoma, immunized with the semi-**allogeneic** hybrid cells, developed CD8-mediated melanoma immunity and survived significantly ($P < 0.005$) longer than mice with melanoma immunized with a mixture of the parental cell types. The failure of melanoma immunity to develop in mice injected with the mixture of parental cells indicated that co-expression of the immunogenic determinants by the same cellular immunogen was necessary for an optimum immunotherapeutic effect. Augmented immunity to melanoma in mice immunized with the semi-**allogeneic** hybrid cells points toward an analogous form of therapy for patients with melanoma.

L134 ANSWER 9 OF 46 CANCERLIT DUPLICATE 13
ACCESSION NUMBER: 83214635 CANCERLIT
DOCUMENT NUMBER: 83214635
TITLE: Augmentation of syngeneic tumor-specific immunity by **semiallogeneic** cell hybrids.
AUTHOR: Toffaletti D L; Darrow T L; Scott D W
CONTRACT NUMBER: CA-22845 (NCI)
T32-GM-07003 (NIGMS)

SOURCE: JOURNAL OF IMMUNOLOGY, (1983). Vol. 130, No. 6, pp. 2982-6.
Journal code: IFB. ISSN: 0022-1767.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Abridged Index Medicus Journals; Priority Journals; Cancer Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 83214635
ENTRY MONTH: 198308

AB Hybrid cell lines were established from fusions between lipopolysaccharide- (LPS) stimulated C57BL/6J spleen cells and MPC-11 tumor cells (45.6TG1.7, abbreviated M45), and were tested for their ability to immunize **semiallogeneic** mice against a parental tumor challenge. These hybrids were tumorigenic in syngeneic (BALB/c X C57BL/6J) F1 (CB6F1) mice but did not grow in **semiallogeneic** (BALB/c X A/J) F1 (CAF1) mice. All hybrids express both parental major histocompatibility antigens (H-2b and H-2d) as detected by indirect immunofluorescence and by their ability to function as either stimulators or targets for allogeneic cytotoxic lymphocytes (CTL). M45 tumor-associated antigens (TAA) were expressed on the hybrid surface as shown by their ability to act as either stimulators or targets for syngeneic CTL specific for M45 TAA. Immunization of **semiallogeneic** CAF1 mice with the hybrids i.p. followed by a challenge with M45 tumor cells resulted in extended survival when compared to untreated mice or animals immunized i.p. with M45 tumor cells. This immunity was specific and was not due to an allogeneic effect; immunization with an unrelated H-2bd tumor, 70Z/3, or H-2bd B6D2F1 spleen cells or with **semiallogeneic** spleen cells plus M45 did not protect mice from M45 challenge. Interestingly, prophylactic priming with **semiallogeneic** hybrid tumor cells or parental myeloma cells led to M45-specific CTL and "help" for an in vitro CTL response; however, the degree of CTL priming by hybrid tumors was not augmented when compared to the level of CTL achieved with parental tumor alone. Hence, stimulation of CTL activity per se by hybrid tumor cells cannot explain the protective effect of hybrid tumor immunization. These studies nevertheless confirm that **semiallogeneic** hybrids, which we show express TAA and alloantigens, can be used to immunize mice against a lethal syngeneic myeloma tumor challenge.

L134 ANSWER 10 OF 46 CANCERLIT
ACCESSION NUMBER: 2000363865 CANCERLIT
DOCUMENT NUMBER: 20363865
TITLE: Fusions of human ovarian carcinoma cells with autologous or **allogeneic** dendritic cells induce antitumor immunity.
AUTHOR: Gong J; Nikrui N; Chen D; Koido S; Wu Z; Tanaka Y; Cannistra S; Avigan D; Kufe D
CORPORATE SOURCE: Dana-Farber Cancer Institute, Massachusetts General Hospital, and Beth Israel/Deaconess Medical Center, Harvard Medical School, Boston, MA 02115, USA.
Gong@dfci.harvard.edu
CONTRACT NUMBER: CA78378 (NCI)
SOURCE: JOURNAL OF IMMUNOLOGY, (2000). Vol. 165, No. 3, pp. 1705-11.
Journal code: IFB. ISSN: 0022-1767.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals; Abridged Index Medicus Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 20363865
ENTRY MONTH: 200009

AB Human ovarian carcinomas express the CA-125, HER2/neu, and MUC1 tumor-associated Ags as potential targets for the induction of active

pecific immunotherapy. In the present studies, human ovarian cancer cells were fused to human dendritic cells (DC) as an alternative strategy to induce immunity against known and unidentified tumor Ags. Fusions of ovarian cancer cells to autologous DC resulted in the formation of heterokaryons that express the CA-125 Ag and DC-derived costimulatory and adhesion molecules. Similar findings were obtained with ovarian cancer cells fused to **allogeneic** DC. The fusion cells were functional in stimulating the proliferation of autologous T cells. The results also demonstrate that fusions of ovarian cancer cells to autologous or **allogeneic** DC induce cytolytic T cell activity and lysis of autologous tumor cells by a MHC class I-restricted mechanism. These findings demonstrate that fusions of ovarian carcinoma cells and DC activate T cell responses against autologous tumor and that the fusions are functional when generated with either autologous or **allogeneic** DC.

L134 ANSWER 11 OF 46 CANCERLIT
ACCESSION NUMBER: 2000534448 CANCERLIT
DOCUMENT NUMBER: 20534448
TITLE: Dendritic cells infected with recombinant fowlpox virus vectors are potent and long-acting stimulators of transgene-specific class I restricted T lymphocyte activity.
AUTHOR: Brown M; Zhang Y; Dermine S; de Wynter E A; Hart C; Kitchener H; Stern P L; Skinner M A; Stacey S N
CORPORATE SOURCE: Cancer Research Campaign Laboratories, Paterson Institute of Cancer Research, Christie Hospital, Manchester, UK.
SOURCE: GENE THERAPY, (2000). Vol. 7, No. 19, pp. 1680-9. Journal code: CCE. ISSN: 0969-7128.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; I
LANGUAGE: English
OTHER SOURCE: MEDLINE 20534448
ENTRY MONTH: 200101

AB The identification of dendritic cells (DC) as the major antigen-presenting cell type of the immune system, combined with the development of procedures for their ex vivo culture, has opened possibilities for tumour immunotherapy based on the transfer of recombinant tumour antigens to DC. It is anticipated that the most effective type of response would be the stimulation of specific, MHC class I restricted cytotoxic T lymphocytes capable of recognising and destroying tumour cells. In order to make this approach possible, methods must be developed for the transfer of recombinant antigen to the DC in such a way that they will initiate an MHC class I restricted response. Here, we demonstrate that murine DC infected with a recombinant fowlpox virus (rFWPV) vector stimulate a powerful, MHC class I restricted response against a recombinant antigen. A rFWPV containing the OVA gene was constructed and used to infect the DC line DC2.4. The infected DC2.4 cells were found to stimulate the T-T cell hybridoma line RF33. 70, which responds specifically to the MHC class I restricted OVA peptide SIINFEKL. The stimulatory ability of the rFWPV-infected DC2.4 cells lasted for at least 72 h after infection and was eventually limited by proliferation of uninfected cells. By comparison, DC2.4 cells pulsed with synthetic SIINFEKL peptide stimulated RF33.70 well initially, but the stimulatory ability had declined to zero by 24 h after pulsing. FWPV infection of DC2.4 up-regulated MHC and costimulatory molecule expression. rFWPV was also found to infect both immature and mature human DC derived from cord blood CD34+ progenitors and express transgenes for up to 20 days after infection. We conclude that rFWPV shows promise as a vector for antigen gene transfer to DC in tumour immunotherapy protocols.

L134 ANSWER 12 OF 46 CANCERLIT
ACCESSION NUMBER: 1999129158 CANCERLIT
DOCUMENT NUMBER: 99129158
TITLE: Comparison of four strategies for tumour vaccination in the B16-F10 melanoma model.
AUTHOR: Souberbielle B E; Westby M; Ganz S; Kayaga J; Mendes R; Morrow W J; Dalgleish A G
CORPORATE SOURCE: Department of Oncology, St George's Hospital Medical School, London, UK.
SOURCE: GENE THERAPY, (1998). Vol. 5, No. 11, pp. 1447-54.
Journal code: CCE. ISSN: 0969-7128.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 99129158
ENTRY MONTH: 199904
AB We have compared four cell-based tumour vaccine strategies in prevention experiments using the B16-F10 melanoma model. Two of these are thought to favour the direct antigen presentation pathway (B16-F10 expressing B7.1 and hybrids made between B16-F10 cells and macrophages) and the other two strategies are thought to act by an indirect pathway of presentation (**allogeneic** tumour cells and autologous tumour cells combined with a powerful adjuvant (Provax-IDEC Pharmaceuticals)). Only the two latter vaccines promoted antitumour activity, whereas the vaccines consisting of B7.1-expressing tumour cells or the hybrid vaccine failed to provide any antitumour activity. Recently human trials have commenced using transfection of the B7.1 molecule, as well as employing the hybrid technology to make tumour-B cell hybrids or tumour and dendritic cell hybrids. Our results suggest that these approaches could be disappointing in the clinics if not optimised.

L134 ANSWER 13 OF 46 CANCERLIT
ACCESSION NUMBER: 96195030 CANCERLIT
DOCUMENT NUMBER: 96195030
TITLE: Interleukin 3 enhances cytotoxic T lymphocyte development and class I major histocompatibility complex "re-presentation" of exogenous antigen by tumor-infiltrating antigen-presenting cells.
AUTHOR: Pulaski B A; Yeh K Y; Shastri N; Maltby K M; Penney D P; Lord E M; Frelinger J G
CORPORATE SOURCE: Department of Microbiology and Immunology, University of Rochester School of Medicine and Dentistry, NY 14642, USA.
CONTRACT NUMBER: CA11198 (NCI)
T32AI07285 (NIAID)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996). Vol. 93, No. 8, pp. 3669-74.
Journal code: PV3. ISSN: 0027-8424.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Cancer Journals; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 96195030
ENTRY MONTH: 199607
AB We show that interleukin 3 (IL-3) enhances the generation of tumor-specific cytotoxic T lymphocytes (CTLs) through the stimulation of host antigen-presenting cells (APCs). The BALB/c (H-2d) spontaneous lung carcinoma line 1 was modified by gene transfection to express ovalbumin as a nominal "tumor antigen" and to secrete IL-3, a cytokine enhancing myeloid development. IL-3-transfected tumor cells are less tumorigenic than the parental cell line, and tumor-infiltrating lymphocytes isolated from these tumors contain increased numbers of tumor-specific CTLs. By

using B3Z86/90.14 (B3Z), a unique T-cell hybridoma system restricted to ovalbumin/H-2b and implanting the tumors in (BALB/c x C57BL/6)F1 (H-2d/b) mice, we demonstrate that the IL-3-transfected tumors contain an increased number of a rare population of host cells that can process and "re-present" tumor antigen to CTLs. Electron microscopy allowed direct visualization of these host APCs, and these studies, along with surface marker phenotyping, indicate that these APCs are macrophage-like. The identification of these cells and their enhancement by IL-3 offers a new opportunity for tumor immunotherapy.

L134 ANSWER 14 OF 46 CANCERLIT
ACCESSION NUMBER: 95167668 CANCERLIT
DOCUMENT NUMBER: 95167668
TITLE: Differences in immune responses to tumor induced in syngeneic hosts by injection of hybrid and parental tumor cells.
AUTHOR: Kambe M; Rou K; Tachibana T
CORPORATE SOURCE: Department of Clinical Oncology, Tohoku University, Sendai.
SOURCE: TOHOKU JOURNAL OF EXPERIMENTAL MEDICINE, (1994). Vol. 174, No. 1, pp. 71-83.
Journal code: VTF. ISSN: 0040-8727.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 95167668
ENTRY MONTH: 199504
AB Immunization of C3H/He mice with L-FM3A#2 hybrid cells, made by fusion of ascitic mammary carcinoma FM3A#2 cells with 8-azaguanine resistant LAG cells, both of C3H/He mouse origin, resulted in spleen T cell-dependent resistance to the parental FM3A/R cells. These spleen T cells, purified by passing through a nylon fiber column, could be demonstrated to have Thy-1.2 and Lyt-2.1 antigens, and not L3/T4 antigens. After immunizing with irradiated FM3A/R cells, cytotoxic cells other than cytotoxic T lymphocytes (CTL) appeared, these presumably being nonphagocytic macrophages or polymorphonuclear cells. In this case, anti MM antiserum was generated at an earlier stage than when mice were immunized with the L-FM3A#2 cells. The cytotoxic mechanism is discussed as to the significance of the surface antigen.

L134 ANSWER 15 OF 46 CANCERLIT
ACCESSION NUMBER: 90056996 CANCERLIT
DOCUMENT NUMBER: 90056996
TITLE: Report of two cases of acute myelogenous leukemia immunized with autologous leukemia-derived hybrid cells.
AUTHOR: Cohen E P; Lazda V A; Schade S G; Kennedy J L; Kaufman E R; Hagen K L
CORPORATE SOURCE: Department of Medicine, University of Illinois College of Medicine, Chicago 60680.
SOURCE: MOLECULAR BIOTHERAPY, (1988). Vol. 1, No. 2, pp. 86-95.
Journal code: AH5. ISSN: 0952-8172.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 90056996
ENTRY MONTH: 199002
AB Reports that immunizations with leukemia-derived hybrid cells prolonged the survival of leukemic mice led us to attempt an analogous approach in two adult patients with acute myeloid leukemia (AML). Hybrid cells were prepared from the pretreatment marrows of the newly-diagnosed patients with D98OR cells, in the first case, and with KR12 cells, in the second case. (D98OR and KR12 cells are human cell-lines.) Hybrids formed with

KR12 cells expressed HLA antigens of both parental sources and some of the clonal isolates expressed myeloid-associated determinants. The immunizations were performed during the first complete clinical remission; the patients were demonstrably immunocompetent. Positive delayed type hypersensitivity responses to both (X-irradiated) hybrid cells and to (X-irradiated) autologous pretreatment marrow were observed following the immunizations. Mixed lymphocyte reactions toward autologous marrow were positive in one of the patients. In both, relapse occurred approximately two months after the first immunization and eight months after first diagnosis. The first patient remained in complete remission for two and one-half years following reinduction chemotherapy; reinduction chemotherapy was unsuccessful in the second patient.

L134 ANSWER 16 OF 46 CANCERLIT
ACCESSION NUMBER: 83155695 CANCERLIT
DOCUMENT NUMBER: 83155695
TITLE: Cytotoxic activity of lymphoid cells from mice immunized with **semiallogeneic** hybrid cells: requirement of in vitro lymphoid cells culture for expression of cytotoxicity against a syngeneic chemically induced tumor.
AUTHOR: Payelle B; Goguel A F; Poupon M F; Lespinats G
SOURCE: CELLULAR IMMUNOLOGY, (1982). Vol. 74, No. 2, pp. 383-93. Journal code: CQ9. ISSN: 0008-8749.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 83155695
ENTRY MONTH: 198306

L134 ANSWER 17 OF 46 CANCERLIT
ACCESSION NUMBER: 82119087 CANCERLIT
DOCUMENT NUMBER: 82119087
TITLE: Serologically defined antigens on the surface of somatic hybrid cells.
AUTHOR: Rubio N
SOURCE: INTERNATIONAL ARCHIVES OF ALLERGY AND APPLIED IMMUNOLOGY, (1982). Vol. 67, No. 2, pp. 123-6. Journal code: GP9. ISSN: 0020-5915.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 82119087
ENTRY MONTH: 198205
AB Y2C somatic cell hybrids, which immunize **semiallogeneic** mice and protect them against further challenge with syngeneic malignant cells, express normal H-2 antigens from parental cells and, in addition, the L antigen and the Friend, Moloney, Rauscher type-specific antigen. This was demonstrated by immunoabsorption, complement-dependent cytotoxicity and immunofluorescence experiments.

L134 ANSWER 18 OF 46 CANCERLIT
ACCESSION NUMBER: 82029803 CANCERLIT
DOCUMENT NUMBER: 82029803
TITLE: Adoptive transfer of immunity induced by **semi-allogeneic** hybrid cells, against a murine fibrosarcoma.
AUTHOR: Payelle B; Poupon M F; Lespinats G
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1981). Vol. 27, No. 6, pp. 783-8. Journal code: GQU. ISSN: 0020-7136.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

.LE SEGMENT: MEDL; L; Priority Journals
LANGUAGE: English
OTHER SOURCE: MEDLINE 82029803
ENTRY MONTH: 198201
AB **Semi-allogeneic** somatic hybrid cells derived from the fusion of a C57BL/6 fibrosarcoma (MCB6-1) and A9 cells (C3H origin) were used to immunize C57BL/6 mice against the parental tumor cells. These hybrid cells expressed H-2 histocompatibility antigen of both parental cells (H-2b and H-2k), and failed to produce tumors in normal C57BL/6 mice. A single i.p. injection of hybrid cells induced anti-tumor immunity which could be transferred to normal C57BL/6 recipient mice by immune spleen or peritoneal cells; the efficient cells were T cells, as this activity was completely abrogated by treatment with anti-Thy-1-2 antiserum and complement. Among immune splenic T cells, only the light-density T cells, obtained after fractionation on Percoll gradient, were effective in the transfer of immunity. Immunity induced by the hybrid cells was specific for MCB6-1 parental tumor cells. This immunity could be transferred during two brief periods, 7 to 12 days, and 40 to 50 days, after hybrid cell injection; there appeared to be an intermediate period, 12 to 40 days after immunization, during which no immunity could be transferred. These results suggest a suppressive mechanism implicated during hybrid cell immunization and interacting with the anti-tumor immune response.

L134 ANSWER 19 OF 46 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 1
ACCESSION NUMBER: 2001:242655 CAPLUS
TITLE: **Antigen-presenting hybridoma** cells expressing **MHC** antigens of the LEW rat
AUTHOR(S): Matsuda, C.; Yokota, A.; Izumi, T.; Shinohara, N.
CORPORATE SOURCE: 1-15-1 Kitasato, Department of Internal Medicine, Kanagawa, Sagamihara, 228-8555, Japan
SOURCE: J. Immunol. Methods (2001), 251(1-2), 93-100
CODEN: JIMMBG; ISSN: 0022-1759
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Towards the eventual purpose of facilitating analyses of specificities and functions of LEW rat T lymphocytes involved in the induction and development of organ-specific autoimmune disorders, **hybridoma** cells expressing class I and class II **MHC** antigens of LEW rat have been developed. B cell **hybridomas** produced between a murine B **cell tumor** M12.4.5 and stimulated LEW B cells expressed high levels of LEW class II **MHC** antigen but the expression of LEW class I **MHC** antigens on these cells was rather low. The B **hybridoma** cells were capable of presenting sol. protein antigens to LEW CD4+ T cells. Furthermore, The use of this **hybridoma** revealed antigen-specific cytolytic activity of rat CD4+ T cells. T cell **hybridomas** produced between murine thymoma BW5147 and LEW T cells expressed class I **MHC** antigens of the LEW rat. The expression was confirmed by surface staining and specific cytolysis by rat **allogeneic** CTL.
REFERENCE COUNT: 23
REFERENCE(S): (1) Eshima, K; Eur J Immunol 1997, V27, P55 CAPLUS
(3) Hanabuchi, S; Proc Natl Acad Sci USA 1994, V91, P4930 CAPLUS
(4) Ishiyama, S; J Immunol 1998, V161, P4695 CAPLUS
(5) Kataoka, T; J Immunol 1996, V156, P3678 CAPLUS
(10) Okura, Y; J Mol Cell Cardiol 1997, V29, P491 CAPLUS
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Searched by Barb O'Bryen, STIC 308-4291

L134 ANSWER 20 OF 46 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 3
ACCESSION NUMBER: 2000:900478 CAPLUS
DOCUMENT NUMBER: 134:46754
TITLE: Use of semi-allogeneic cell line-peptide complexes for the treatment of cancer, AIDS and other viral diseases
INVENTOR(S): Gattoni-celli, Sebastiano; Shearer, Gene; Grene, Edith; Newton, Danforth A.; Brown, Edwin A.; Berzofsky, Jay A.; Degroot, Anne S.
PATENT ASSIGNEE(S): The Government of the United States of America, as Represented by the Secret, USA; Medical University of South Carolina
SOURCE: PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000076537	A2	20001221	WO 2000-US11008	20000424
WO 2000076537	A3	20010503		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
WO 9811202	A1	19980319	WO 1997-US15920	19970910
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: WO 1997-US15920 A2 19970910
US 1999-254556 A2 19990616
US 1996-707920 A2 19960910

AB The present invention provides a compn. comprising a semi-allogeneic hybrid fusion cell and an immunogenic peptide. In particular, isolated peptides of HIV (Human Immunodeficiency Virus), HTLV-1, Hepatitis B virus, Hepatitis C virus, rubeola virus, influenza A virus and Human Papilloma Virus are provided in the compns. of the present invention. Moreover, isolated cancer-specific peptides specific to a cancer, for example, B cell lymphoma, T cell lymphoma, myeloma, leukemia, breast cancer, pancreatic cancer, colon cancer, lung cancer, renal cancer, liver cancer, prostate cancer, melanoma and cervical cancer are provided in the compns. of the present invention. Moreover, the present invention provides a method of treating a subject infected by one or more of HIV, HTLV-1, Hepatitis B virus, Hepatitis C virus, rubeola virus, influenza A virus and Human Papilloma Virus, comprising administering a compn. comprising an effective amt. of a hybrid fusion cell and an effective amt. of an isolated immunogenic peptide of the virus in a pharmaceutically acceptable carrier. Further, the present invention provides a method of treating cancer in a subject with one or more of B cell lymphoma, T cell lymphoma,

myeloma, leukemia, breast cancer, pancreatic cancer, colon cancer, lung cancer, renal cancer, liver cancer, prostate cancer, melanoma and cervical cancer, comprising administering a compn. comprising an effective amt. of a hybrid fusion cell and an effective amt. of an isolated immunogenic peptide of the cancer in a pharmaceutically acceptable carrier.

L134 ANSWER 21 OF 46 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 9
ACCESSION NUMBER: 1998:542991 CAPLUS
DOCUMENT NUMBER: 129:160641
TITLE: Cancer immunotherapy with **semi-allogeneic** cells
INVENTOR(S): Cohen, Edward P.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9833527	A2	19980806	WO 1998-US1824	19980130
WO 9833527	A3	19981105		
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1012240	A2	20000628	EP 1998-904782	19980130
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, NL, IE				
US 6187307	B1	20010213	US 1998-16528	19980130

PRIORITY APPLN. INFO.: US 1997-36620 P 19970131
WO 1998-US1824 W 19980130

AB The present invention relates to improved **semi-allogeneic** immunogenic cells which act to stimulate and induce an immunol. response when administered to an individual. In particular, it relates to cells which express both **allogeneic** and syngeneic **MHC** determinants and which also express at least one antigen recognized by T lymphocytes. The invention is also directed to methods of inducing an immune response and methods of treating tumors by administering the **semi-allogeneic** immunogenic cells to an individual.

L134 ANSWER 22 OF 46 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 10
ACCESSION NUMBER: 1998:183995 CAPLUS
DOCUMENT NUMBER: 128:242890
TITLE: Semi-allogeneic cell hybrids as preventive and therapeutic vaccines for cancer and AIDS
INVENTOR(S): Gattoni-Celli, Sabastiano; Newton, Danforth A.; McClay, Edward F.
PATENT ASSIGNEE(S): Medical University of South Carolina, USA; Gattoni-Celli, Sabastiano; Newton, Danforth A.; McClay, Edward F.
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9811202	A1	19980319	WO 1997-US15920	19970910

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6063375	A	20000516	US 1996-707920	19960910
AU 9743382	A1	19980402	AU 1997-43382	19970910
EP 927244	A1	19990707	EP 1997-941483	19970910

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

JP 2001500731	T2	20010123	JP 1998-513774	19970910
WO 2000076537	A2	20001221	WO 2000-US11008	20000424
WO 2000076537	A3	20010503		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:	US 1996-707920	A2 19960910
	WO 1997-US15920	W 19970910
	US 1999-254556	A2 19990616

AB An isolated cell or cell line, wherein the cell is .beta.2-microglobulin deficient, neomycin-resistant and HAT-sensitive is provided. The cell FO-1 #12 is an example of a cell having these characteristics. A cell hybrid formed by the fusion of an FO-1 #12 cell or other cell described herein and a mammalian cell is provided. The patient-derived cell can be a tumor cell or other cell, such as a white blood cell. The patient-derived tumor cell can be a melanoma cell, a prostatic carcinoma cell, a colon carcinoma cell, a lung carcinoma cell, a breast carcinoma cell, a pancreatic carcinoma cell, or others. A method of treating AIDS in a patient, comprising administering to the patient a cell hybrid provided herein, wherein the patient-derived white blood cell is derived from the patient being treated, is provided. A method of treating solid tumor in a patient, comprising administering to the patient a cell hybrid as provided herein, wherein the patient-derived tumor cell is derived from the patient being treated, is provided.

L134 ANSWER 23 OF 46 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER:	2001:287195 CAPLUS
TITLE:	Genetically modified **tumour** vaccines: an obstacle race to break host tolerance to cancer
AUTHOR(S):	Nawrocki, Sergiusz; Wysocki, Piotr J.; Mackiewicz, Andrzej
CORPORATE SOURCE:	Department of Radiation Oncology & Department of Cancer Immunology, USOMS, GreatPoland Cancer Centre, Poznan, 61-866, Pol.
SOURCE:	Expert Opin. Biol. Ther. (2001), 1(2), 193-204 CODEN: EOBTA2; ISSN: 1471-2598
PUBLISHER:	Ashley Publications Ltd.
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB The development of genetically modified tumor vaccines (GMTV) has been prompted by a better understanding of antitumor immune responses and genetic engineering technologies, as well as the identification of

numerous tumor antigens (TA) in several malignancies which occasionally induce spontaneous tumor regressions. Cellular vaccines are based on autologous or **allogeneic tumor cells** genetically engineered to secrete different cytokines, co-stimulatory mols., or **allogeneic HLA** mols. in order to provide a strong stimulatory signal together with the presented TA. Another promising approach that is targeted towards breaking immune tolerance to TA, exploits **dendritic** cells (DC) loaded or genetically modified with TA (and sometimes cytokines). Effective nonviral and viral gene delivery systems have been constructed including a third generation of adenoviral, lentiviral and **hybrid** vectors. Studies in mice demonstrated that therapeutic, curative immune responses might be elicited by GMTV. Promising results from animal studies are rarely seen in human trials. Several reasons, such as numerous escape mechanisms of slowly evolving spontaneous tumors and immune incompetence of advanced patients, are major concerns. Improved monitoring of immune responses to GMTV is essential to distinguish between responders and non-responders in order to tailor immune therapy strategy to the individual patient.

REFERENCE COUNT: 75

REFERENCE(S):
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(2) Azuma, M; Curr Top Microbiol Immunol 1995, V198, P59 CAPLUS
(3) Bakker, A; J Exp Med 1994, V179, P1005 CAPLUS
(4) Bakker, A; J Immunol 1998, V160, P5239 CAPLUS
(5) Banchereau, J; Nature 1998, V392, P245 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L134 ANSWER 24 OF 46 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:718245 CAPLUS
DOCUMENT NUMBER: 133:295356
TITLE: **Fusion** proteins of novel CTLA4/CD28 ligands and uses therefore
INVENTOR(S): Freeman, Gordon J.; Nadler, Lee M.; Gray, Gary S.; Greenfield, Edward
PATENT ASSIGNEE(S): Dana Farber Cancer Institute, USA; Replingen Corporation
SOURCE: U.S., 83 pp., Cont.-in-part of U.S. Ser. No. 109,393, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6130316	A	20001010	US 1994-280757	19940726
US 5942607	A	19990824	US 1993-101624	19930726
CA 2167091	AA	19950202	CA 1994-2167091	19940726
US 6084067	A	20000704	US 1995-479744	19950607
AU 9896991	A1	19990218	AU 1998-96991	19981208

PRIORITY APPLN. INFO.:
US 1993-101624 B2 19930726
US 1993-109393 B2 19930819
US 1993-147773 B2 19931103
AU 1994-74052 A3 19940726
US 1994-280757 A2 19940726
WO 1994-US8423 W 19940726

AB Nucleic acids encoding novel CTLA4/CD28 ligands which costimulate T cell activation are disclosed. In one embodiment, the nucleic acid has a sequence which encodes a B lymphocyte antigen, B7-2. Preferably, the

nucleic acid is a DNA mol. comprising at least a portion of a nucleotide sequence shown in FIG. 8, SEQ ID NO:1 or FIG. 14, SEQ ID NO:23. The nucleic acid sequences of the invention can be integrated into various expression vectors, which in turn direct the synthesis of the corresponding proteins or peptides in a variety of hosts, particularly eukaryotic cells, such as mammalian and insect cell culture. Also disclosed are host cells transformed to produce proteins or peptides encoded by the nucleic acid sequences of the invention and isolated proteins and peptides which comprise at least a portion of a novel B lymphocyte antigen. Proteins and peptides described herein can be administered to subjects to enhance or suppress T cell-mediated immune responses.

REFERENCE COUNT: 17

REFERENCE(S):
(1) Anon; WO 93/00431 1993 CAPLUS
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(3) Capon; US 5116964 1992 CAPLUS
(4) Freedman, A; Journal of Immunology 1987, V139(10), P3260 CAPLUS
(5) Freeman, G; Journal of Experimental Medicine 1991, V174, P625 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L134 ANSWER 25 OF 46 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1999:168743 CAPLUS
DOCUMENT NUMBER: 131:17735

TITLE: Monoclonal IgG antibodies influence the migration patterns of lymphocytes in vivo

AUTHOR(S): Yousaf, Nasim; Williams, Bryan D.
CORPORATE SOURCE: Department of Rheumatology, University of Wales College of Medicine, Cardiff, CF4 4XN, UK

SOURCE: Int. Arch. Allergy Immunol. (1999), 118(1), 59-66
CODEN: IAAIEG; ISSN: 1018-2438
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Monoclonal antibodies (MoAb) are useful therapeutic agents for the treatment of a variety of human disorders, although the effector mechanisms responsible for the outcome of an efficient immunotherapy remain unclear. This study was designed to address the early effects of MoAb on the migration patterns of lymphocytes in vivo. The clearance profiles and tissue distribution of 111In-labeled rat lymph node cells were examd. in both normal and decomplemented allogeneic and **semi-allogeneic** recipients pre-injected with IgG2b (R3/13) or IgG2a (R2/15S) MoAb directed against the RT1Aa, the classical class I major histocompatibility complex antigen of the DA rat. Both MoAb were equally effective in not only augmenting the removal of DA and (DA .times. PVG)F1 cells from the circulation and promoting their subsequent localization within the liver but also causing cell lysis during the early phase of cell clearance, even in decomplemented recipients. Although R3/13 and R2/15S are known to target erythrocytes differently in normal and cobra venom factor (CVF)-treated animals, no differences were obsd. in the migration behavior of lymph node cells in allogeneic or **semi-allogeneic** hosts pre-injected with the same MoAb. Since rat lymphocytes express a much higher level of the RT1Aa antigen as compared with erythrocytes, the authors could not exclude a possible role of residual complement components in the circulation of CVF-treated rats that may have accounted for the obsd. antibody-dependent effects on target lymphocytes. It is believed that the design and methodol. employed in the authors' present exptl. opsonization system were inadequate to define clearly the mechanisms responsible for antibody-mediated removal and

destruction of target lymphocytes in vivo.
REFERENCE COUNT: 48
REFERENCE(S): (1) Aase, A; Scand J Immunol 1994, V39, P581 CAPLUS
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(10) Cox, J; Transplantation 1984, V38, P17 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L134 ANSWER 26 OF 46 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:689486 CAPLUS
DOCUMENT NUMBER: 125:317348
TITLE: **Dendritic**-like cell **fusion** with immortal **tumor cell** line to form **hybrids** and **hybridomas** for cancer patient immunization and stimulation of anti-tumor response
INVENTOR(S): Moser, Muriel; Leo, Oberdan; Lespagnard, Laurence; Urbain, Jacques; Bruyns, Catherine; Gerard, Catherine; Goldman, Michel; Velu, Thierry; Willems, Fabienne; et al.
PATENT ASSIGNEE(S): Baxter International Inc., USA
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9630030	A1	19961003	WO 1996-US4370	19960329
W: AU, CA, CN, JP, KR, SG				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9654367	A1	19961016	AU 1996-54367	19960329
EP 839044	A1	19980506	EP 1996-911493	19960329
R: BE, CH, DE, FR, GB, LI, NL				

PRIORITY APPLN. INFO.: US 1995-414480 19950331
WO 1996-US4370 19960329

AB The invention provides **dendritic**-like **cell/tumor cell hybridomas** and pluralities of **dendritic**-like **cell/tumor cell hybrids** that confer tumor resistance in vivo. The **hybrids** and **hybridomas** are generated by the fusion of **tumor cells** with **dendritic**-like cells. For instance, immortal **tumor cells** from an autologous **tumor cell** line can be fused with autologous or **HLA**-matched **allogeneic dendritic**-like cells. Autologous **tumor cell** lines can be drived from primary tumors and from their metastases. Alternatively, immortal **dendritic**-like cells from an autologous or **allogeneic HLA**-matched **dendritic**-like cell line can be fused with autologous **tumor cells**. Autologous **dendritic**-like cell lines can be prepd. from various sources such as peripheral blood and bone marrow. **Dendritic**-like **cell/tumor cell hybridomas** and pluralities of **hybrids** can be directly infused for active immunization of cancer patients against their residual **tumor cells**. The **hybridomas** and **hybrids** can also be used for the in vitro activation of autologous immune cells before their reinfusion into the patient for passive immunization against the **tumor cells**.

L134 ANSWER 27 OF 46 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:539213 BIOSIS
DOCUMENT NUMBER: PREV200000539213
TITLE: **Hybrid** cell vaccination of autologous **tumor** cells **fused** with **allogeneic** **dendritic** cells as **therapy** in progressive metastatic renal cell carcinoma (RCC.
AUTHOR(S): Becker, V. (1); Strutz, F. (1); Kulger, A.; Ringert, R. H.; Fenner, W.; Schott, W.; Mueller, C. A.; Mueller, G. A. (1)
CORPORATE SOURCE: (1) Department of Nephrology and Rheumatology, University of Goettingen, Goettingen Germany
SOURCE: Kidney & Blood Pressure Research, (2000) Vol. 23, No. 3-5, pp. 277. print.
Meeting Info.: Congress of Nephrology 2000 Vienna, Austria September 02-05, 2000 Gesellschaft fuer Nephrologie . ISSN: 1420-4096.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L134 ANSWER 28 OF 46 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1999:445201 BIOSIS
DOCUMENT NUMBER: PREV199900445201
TITLE: Calcium signaling induces acquisition of **dendritic** cell characteristics in chronic myelogenous leukemia myeloid progenitor cells.
AUTHOR(S): Engels, Friederike H. C.; Koski, Gary K.; Bedrosian, Isabelle; Xu, Shuwen; Luger, Selina; Nowell, Peter C.; Cohen, Peter A.; Czerniecki, Brian J. (1)
CORPORATE SOURCE: (1) Department of Surgery, University of Pennsylvania Medical Center, 3400 Spruce Street, 4 Silverstein Pavilion, Philadelphia, PA, 19104-4283 USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (Aug. 31, 1999) Vol. 96, No. 18, pp. 10332-10337.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Effective host T lymphocyte sensitization to malignant cells depends on successful **antigen presentation**. In this study, we examined the capacity of malignant myeloid progenitor cells of patients in the chronic phase of chronic myelogenous leukemia (CML) to acquire characteristics of activated **dendritic** cells (DCs) after intracellular calcium mobilization, thereby bypassing a need for third-party **antigen-presenting** cells. Treatment of purified CD33+ CML cells from 15 patients with calcium ionophore (CI) consistently resulted in de novo expression of the costimulatory molecules CD80 (B7.1) and CD86 (B7.2), CD40 and the DC-specific activation marker CD83, as well as marked up-regulation of **MHC** class I and II molecules and the adhesion molecule CD54. Most of these changes occurred within 24 hr of treatment. Morphologically, CI-treated CML cells developed long **dendritic** projections similar to those seen in mature DCs. Functionally, CI-treated CML cells provided stimulation of **allogeneic** Tlymphocytes 10- to 20-fold that of untreated CML cells or untreated monocytes. Fluorescent in situ **hybridization** of CI-activated CML cells confirmed their leukemic origin by displaying the typical bcr/abl **fusion** signal. No difference in bcr/abl translocation percentages between untreated and CI-treated CML nuclei was observed. These observations indicate that calcium mobilization may

constitute a valuable approach for rapidly and reliably generating CML-derived DCs for **immunotherapy** of CML.

L134 ANSWER 29 OF 46 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:23931 BIOSIS
DOCUMENT NUMBER: PREV200000023931
TITLE: Clonal heterogeneity of **dendritic** cells derived from patients with chronic myeloid leukemia and enhancement of their T-cells stimulatory activity by IFN-alpha.
AUTHOR(S): Wang, Chun; Al-Omar, Hamad M.; Radvanyi, Laszlo; Banerjee, Avik; Bouman, Derek; Squire, Jeremy; Messner, Hans A. (1)
CORPORATE SOURCE: (1) Princess Margaret Hospital/Ontario Cancer Institute, 610 University Ave., Toronto, ON, M5G 2M9 Canada
SOURCE: Experimental Hematology (Charlottesville), (July, 1999) Vol. 27, No. 7, pp. 1176-1184.
ISSN: 0301-472X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Adoptive **immunotherapy** in form of donor leukocyte infusions is effective in a significant number of patients with chronic myeloid leukemia (CML) that have relapsed after **allogeneic** bone marrow transplantation (BMT). However, the therapy is associated with clinically significant side effects such as graft-versus-host disease (GVHD) and bone marrow (BM) hypoplasia that may be avoided through the administration of T cells with specific antileukemic activity. **Dendritic** cells (DC) functioning as potent **antigen presenting** cells (APC) may play an important role in the generation of T cells with specificity against CML. We examined a subpopulation of CD1a+/CD14- DC generated in vitro from BM of normal subjects and patients with CML using granulocyte-macrophage colony-stimulating factor (GM-CSF), **tumor** necrosis factor-alpha (TNF-alpha) and interleukin-4 (IL-4). These DC derived from both the BM of normal subjects and of patients with CML, differentiated and matured in culture in a similar way. Howe ver, DC derived from patients with CML, displayed decreased activity when tested with **allogeneic** T cells in a mixed lymphocyte reaction (MLR). Addition of interferon-alpha (IFN-alpha) to DC cultures significantly upregulated the expression of major **histocompatibility** complex (**MHC**) molecules (class I and class II) and costimulatory molecules (B7.1 and B7.2) on DC from normal donors and CML patients. However, DC grown from CML patients required a higher concentration of IFN-alpha. IFN-alpha also significantly improved the capacity of CML DC to stimulate T-lymphocyte responses. Fluorescence in situ **hybridization** (FISH) showed that only some CD1a+/CD14- DC derived from BM of patients with CML expressed the bcr/abl **fusion** gene. Incubation with INF-alpha decreased the proportion of bcr/abl positive DC.

L134 ANSWER 30 OF 46 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:527586 BIOSIS
DOCUMENT NUMBER: PREV200000527586
TITLE: **Hybrid** cell vaccine of autologous **tumor** cells and **allogeneic dendritic** cells for active specific immune **therapy** of progressive metastatic renal cell carcinoma (RCC.
AUTHOR(S): Becker, V. (1); Berner, B. (1); Strutz, F. (1); Kugler, A.; Kallerhoff, M.; Thelen, P.; Fenner, W.; Schott, W.; Ringert, R. H.; Mueller, C. A.; Mueller, G. A. (1)
CORPORATE SOURCE: (1) Department of Nephrology and Rheumatology, University of Goettingen, Goettingen Germany
SOURCE: Kidney & Blood Pressure Research, (1999) Vol. 22, No. 4-6, pp. 255. print.

Meeting Info.: Joint Scientific Meeting of the Society for Nephrology and the German Working Group for Clinical Nephrology Freiburg, Germany September 18-21, 1999
ISSN: 1420-4096.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L134 ANSWER 31 OF 46 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1983:193048 BIOSIS
DOCUMENT NUMBER: BA75:43048
TITLE: **TUMORIGENICITY** AND **TUMOR** GRAFT REJECTION OF POLYOMA VIRUS TRANSFORMED FIBROBLAST T LYMPHOCYTE **HYBRIDS**.
AUTHOR(S): FOA C; BEREBBI M; LIPCEY C; GALINDO J R; BONNEAU H
CORPORATE SOURCE: UNITE RECHERCHES CANCEROL. EXPERIMENTALE, U. 119 INSERM, 27, BD LEI ROURE, 13009, MARSEILLE, FRANCE.
SOURCE: BR J EXP PATHOL, (1982) 63 (3), 305-314.
CODEN: BJEPA5. ISSN: 0007-1021.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB In anticipation of the use of functional T-lymphocyte **hybrids** in adoptive **immunotherapy**, the differentiation and **tumorigenicity** of **hybrid** clones generated by **fusion** of a T lymphocyte derived from F1 (DBA/J2 .times. AKR) mouse spleen and a polyoma virus-transformed fibroblast initiated from C3H mouse cells were studied. The **hybrid** cells grew in suspension and had an appearance (by transmission and scanning EM) very similar to that of the lymphocytic line. The **hybrid** and the different clones could induce **tumor** grafts. Malignancy was dominant in newborn mice where **tumors** were obtained in all mouse strains (allogeneic or **semiallogeneic**) inoculated. In adult mice, the **hybrid** cells were **tumorigenic** in C3H and F1 (DBA/J2 .times. AKR), there was complete **tumor** rejection in allogeneic (C57/BL6) or **semi-allogeneic** (DBA/J2 and AKR) mice. The role played by major histocompatibility antigens in the graft rejection is discussed. The histology of the **tumor** grafts was intermediate between fibrosarcoma and lymphosarcoma.

L134 ANSWER 32 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93031900 EMBASE
DOCUMENT NUMBER: 1993031900
TITLE: H-2 I-E molecules isolated from Mls1a stimulatory cells do not activate Mls1a-responsive T cells but do present exogenous staphylococcal enterotoxins.
AUTHOR: MacPhail S.; Stutman O.
CORPORATE SOURCE: Department of Surgery, North Shore University Hospital, Research Building, 300 Community Drive,Manhasset, NY 11030, United States
SOURCE: European Journal of Immunology, (1993) 23/1 (90-95).
ISSN: 0014-2980 CODEN: EJIMAF
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The T cell response to **allogeneic** murine Mls determinants is not H-2 restricted but is dependent on H-2 class II molecules on the Mls-expressing stimulator cells. We have tested planar membranes containing H-2 class II I-E molecules alone or with I-A molecules for their ability to activate a panel of Mls1a-specific T hybrids. Despite the

ability of the planar membranes to activate an alloreactive T hybrid and to present staphylococcal enterotoxins or an antigenic peptide to appropriately responsive T hybrids, they failed to stimulate the Mls1a-specific T hybrids. These findings, in the light of the various controls demonstrating sufficiency of the I-E molecules in the planar membranes, indicate that Mls1a determinants are not covalently bound to I-E molecules; the two molecular species are thus either not physically associated or are linked by a relatively weak interaction. In addition, our experiments show that isolated I-E molecules but not I-A molecules present staphylococcal enterotoxins A and B to two independently derived T hybrids expressing T cell receptor V.beta.1,V.beta.8.2 and V.beta.6 elements.

L134 ANSWER 33 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 92106240 EMBASE
DOCUMENT NUMBER: 1992106240
TITLE: Generation of human IgG, IgA, and IgM anti-melanoma monoclonal antibodies utilizing lymphocytes of an actively immunized melanoma patient.
AUTHOR: Abdel-wahab Z.A.; Gillanders W.E.; Darrow T.L.; Seigler H.F.
CORPORATE SOURCE: Duke University Medical Center, Box 3966, Durham, NC 27710, United States
SOURCE: Human Antibodies and Hybridomas, (1992) 3/1 (32-39). ISSN: 0956-960X CODEN: HANHEX
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Active specific immunotherapy with irradiated **allogeneic** melanoma cells has been shown to enhance the humoral immune response in melanoma patients. An increased titer of melanoma-binding antibodies was demonstrated in sera of immunized patients. Lymph node cells and splenocytes isolated from an actively immunized melanoma patient were fused with the human-murine heteromyeloma cell lines SHMD-33, SPM4-0, and SBC-H20. A group of human anti- melanoma monoclonal antibodies (MABs) were generated from the SHMD-33 fusion. Isolated MABs (one IgG2, one IgA, and two IgM) have been stable in cultures for more than 12 months and have produced human immunoglobulins at 0.2-0.9 Ug/ml/day. As shown by solid phase radioimmunoassays, the MABs react with autologous tumor cells and **allogeneic** melanoma tumors, including the cell line that was used for immunotherapy. In immunocytochemical assays, all four MABs react with a number of melanoma tumor cell lines. The IgG2 and IgA MABs stained preferentially melanoma tumor cells. In contrast, the IgM MABs crossreacted with a broad panel of tumor cells from colon, prostate, pancreas, lung, and other human tumors. The MABs appear to be directed to intracellular rather than membrane-associated antigens as shown by immunofluorescence assays on live and permeabilized cells. The IgG2 antibody recognizes a 70 kDa antigen in melanoma cell lysates by Western immunoblotting. The target antigens for the other MABs have not yet been defined. Stability in culture and strong binding to melanoma tumor cells provide the basis for evaluating the potential of these human MABs. The IgG2 MAB, in particular, may prove useful for diagnostic and therapeutic applications in humans. This study emphasizes the efficacy of primed B lymphocytes isolated from immunized patients for the generation of human MABs of different isotypes.

L134 ANSWER 34 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 88174419 EMBASE

DOCUMENT NUMBER: 1988174419
TITLE: Cytolysis by cloned helper T cells: Induction by specific antigen or by anti-CD3 hybrid antibodies.
AUTHOR: Vyakarnam A.; Strangeways A.L.; Glover R.E.; Lachmann P.J.
CORPORATE SOURCE: Mechanisms in Tumour Immunity Unit, MRC Centre, Cambridge CB2 2QH, United Kingdom
SOURCE: Scandinavian Journal of Immunology, (1988) 27/6 (635-644). ISSN: 0300-9475 CODEN: SJIMAX
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We have explored the use of hybrid antibodies - prepared by covalently linking anti-CD3 to an antibody specific for a monomorphic major histocompatibility complex (MHC) class II determinant using N-succinimidyl 3-(2-pyridyldithio)proprionate/succinimidyl 4-(N-malcimidomethyl)cyclohexane-1-carboxylate (SPDP/SMCC) as coupling reagent - in inducing cytolysis in human tuberculin (PPD)-specific T helper (T(H)) clones. These clones have been shown to lyse PPD-bound Epstein-Barr virus (EBV)-transformed B-cell lines (B-EBV) in an MHC Class II-restricted manner. In this paper anti-CD4-induced cytolysis is compared with antigen/MHC-induced cytolysis with the same clones. Cytolysis induced by the hybrid antibodies was highly efficient, with killing of both syngeneic and **allogeneic** tumour cells positive for MHC class II. Conjugate-induced cytolysis was maximal within 4 h; that of antigen-positive targets at 16 h. Killing of bystander cells was seen only when cytolysis was triggered by antigen/MHC, suggesting that the mechanism of cytolysis in the two systems may be distinct. Targets treated simultaneously with hybrid antibody and with antigen, thereby providing both activation signals to the clones, are lysed less efficiently than those treated with either PPD or hybrid antibody alone. Evidence is presented showing that this inhibition is most marked against syngeneic PPD-coated cells treated with hybrid antibody, suggesting that two signals independently capable of activating cytolytic function in the clones, when presented simultaneously, interfere with the induction of the cytolytic process.

L134 ANSWER 35 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 87085402 EMBASE
DOCUMENT NUMBER: 1987085402
TITLE: Arsonate-specific murine T cell clones. IV. Properties of I-E- and I-A-restricted clones.
AUTHOR: Spragg J.H.; Goodman J.W.
CORPORATE SOURCE: Department of Microbiology and Immunology, University of California San Francisco, San Francisco, CA 94143, United States
SOURCE: Journal of Immunology, (1987) 138/4 (1169-1177). CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English

AB The T cell antigen L-tyrosine-p-azobenzenearsonate is unique in being a simple determinant that can be presented in the context of both I-A and I-E. I-E-restricted T cell clones derived from B10.A(5R) mice were found to fall into three groups: Type I clones recognized antigen only in the context of syngeneic apcs, Type II clones recognized antigen with the same highly specific major histocompatibility complex restriction but in

addition proliferated in response to **allogeneic** stimuli; Type III clones were 'degenerate' in their major histocompatibility complex-restricted recognition of antigen and proliferated when antigen-presenting cells bearing E(.beta.)bE(.alpha.)(k) (syngeneic), E(.beta.)(k)E(.alpha.)(k), or E(.beta.)(d)E(.alpha.)(d) were used. These observations allow some conclusions to be drawn about sites on the I-E molecule that may be functionally significant in the presentation of this antigen. By using the B cell hybridoma LK35.2 as target cells, some of these T cell clones act as cytotoxic cells in the Class II-restricted manner predicted from the results of proliferative assays. Class II-restricted cytotoxicity can therefore be controlled by both I-A and I-E mouse Ir gene loci.

L134 ANSWER 36 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 87008451 EMBASE
DOCUMENT NUMBER: 1987008451
TITLE: The regulatory role of sialic acids in the response of class II reactive T cell hybridomas to **allogeneic** B cells.
AUTHOR: Taiara S.; Kakiuchi T.; Minami M.; Nariuchi H.
CORPORATE SOURCE: Department of Allergology, Institute of Medical Science, University of Tokyo, 108 Tokyo, Japan
SOURCE: Journal of Immunology, (1986) 137/8 (2448-2454). CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
025 Hematology
LANGUAGE: English
AB Two different kinds of alloreactive T cell hybridomas were established in previous experiments. One is reactive and the other is nonreactive to **allogeneic** I-A region-associated membrane antigen (mIa) on B cells. In the present experiments the difference between these hybridomas were analyzed by using representative clones, B cell mIa-reactive clone CB-11.4, and nonreactive clone HTB-9.3. Unresponsiveness of HTB-9.3 clone to **allogeneic** B cells could not be due to the inability of B cells in interleukin 1 production or the density of mIa molecules on B cells. HTB-9.3 clone could respond to C57BL/6 mouse B cells treated with neuraminidase (Nase), and Nase-treated HTB-9.3 clone could respond to normal B cells from C57BL/6 mouse, indicating that sialic acid on both B cells and HTB-9.3 clone plays a regulatory role in the alloreactivity of the clone. In response to B cells from C57BL/6 mouse, T cells from C3H/He mouse spleen showed similar reactivity to HTB-9.3 clone; that is, T cells could respond to Nase-treated B cells, and Nase-treated T cells to B cells, and T cells primed with C57BL/6 spleen cells in vitro showed similar reactivity to CB-11.4 clone. These results suggest that HTB-9.3 clone represents virgin T cells and CB-11.4 clone-primed T cells at least in alloreactivity. Anti-L3T4a was shown to block alloreactivities of both T cell hybridomas and splenic T cells against B cells more efficiently than against splenic adherent cells. These results suggest that L3T4a on T cell plays more important role in **allogeneic** response to B cells than to splenic adherent cells.

L134 ANSWER 37 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 85184380 EMBASE
DOCUMENT NUMBER: 1985184380
TITLE: Functional analysis of cloned macrophage hybridomas. IV. Induction and inhibition of mixed lymphocyte responses.
AUTHOR: Ju S.T.; Dorf M.E.
CORPORATE SOURCE: Harvard Medical School, Department of Pathology, Boston, MA 02115, United States

SOURCE: Journal of Immunology, (1985) 134/6 (3722-3730).
CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
025 Hematology
016 Cancer
LANGUAGE: English

AB A series of macrophage (M.diameter.) hybridomas were generated by fusion of drug-marked P388D1 (H-2(d)) tumor cells by CKB (H-2(k)) splenic adherent cells. The ability of this panel of cloned M.diameter. hybridomas expressing various levels of surface Ia antigens to induce **allogeneic** mixed lymphocytes responses (MLR) was examined. All MLR stimulatory M.diameter. hybridomas expressed surface Ia antigens. However, some Ia+ and all Ia- M.diameter. hybridomas were unable to induce vigorous MLR responses. Furthermore, even after induction of surface Ia antigen expression with Con A supernatants (Con A Sn) or purified interferon-.gamma., the nonstimulatory M.diameter. hybridomas remained ineffective at inducing strong MLR proliferative responses. Furthermore, addition of the latter M.diameter. hybridoma clones (both with and without Con A Sn treatment) to conventional MLR cultures resulted in inhibition of MLR responses. The series of inhibitory M.diameter. hybridomas secreted normal levels of IL 1 upon stimulation with lipopolysaccharide. After surface Ia induction with Con A Sn, the inhibitory M.diameter. hybridomas could stimulate secretion of IL 2 and expression of IL 2 receptors. Moreover, although they inhibited conventional MLR responses, IL 2 production and IL 2 receptor expression were not significantly inhibited. Addition of these M.diameter. hybridomas 24 to 48 hr after initiation of MLR response also inhibited MLR proliferation. The results indicated that the group of inhibitory M.diameter. hybridomas can inhibit MLR responses after IL 2 secretion and acquisition of IL 2 receptors. Finally, this inhibitory activity has been maintained during 1 yr of continuous in vitro culture, and the hybridomas represent a stable 'homogeneous' subpopulation of inhibitory macrophages. Thus, the inhibitory phenotype appears to reflect arrest at a distinct differentiation stage.

L134 ANSWER 38 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 86014600 EMBASE
DOCUMENT NUMBER: 1986014600
TITLE: Qualitative and quantitative studies of antigen-presenting cell function by using I-A-expressing L cells.
AUTHOR: Lechler R.I.; Norcross M.A.; Germain R.N.
CORPORATE SOURCE: Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, United States
SOURCE: Journal of Immunology, (1985) 135/5 (2914-2922).
CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English

AB I-A-expressing transfected murine L cells were analyzed as model antigen-presenting cells. Four features of accessory cell funtion were explored: antigen processing, interaction with accessory molecules (LFA-1, L3T4), influence of Ia density, and ability to stimulate resting, unprimed T lymphocytes. I-A+ L cells could present complex protein antigens to a variety of T cell hybridomas and clones. Paraformaldehyde fixation before but not subsequent to antigen exposure rendered I-A+ L cells unable to present intact antigen. These results are consistent with earlier studies that made use of these methods to inhibit 'processing' by conventional antigen-presenting cells. The ability of anti-L3T4 antibody to inhibit T

cell activation was the same for either B lymphoma or L cell antigen-presenting cells. In striking contrast, anti-LFA-1 antibody, which totally blocked B lymphoma-induced responses, had no effect on L cell antigen presentation, measured as interleukin 2 (IL 2) release by T hybridomas, proliferation, IL 2 release, or IL 2 receptor upregulation by a T cell clone. I-A+ L cell transfectants were found to have a stable level of membrane I-A and I-A mRNA, even after exposure to interferon-.gamma.-containing T cell supernatants. In agreement with earlier reports, a proportional relationship between the (Ia) x (Ag) product and T cell response was found for medium or bright I-A+ cells. However, dull I-A+ cells had a disproportionately low stimulatory capacity, suggesting that there may be a threshold density of Ia per antigen-presenting cell necessary for effective T cell stimulation. Finally, I-A-bearing L cells were shown to trigger low, but reproducible primary **allogeneic** mixed lymphocyte responses with the use of purified responder T cells, indicating that they are capable of triggering even resting T cells. These studies confirm the importance of antigen processing and I-A density in antigen-presenting cell function, but raise questions about the postulated role of the LFA-1 accessory molecule in T cell-antigen-presenting cell interaction. They also illustrate the utility of the L cell transfection model for analysis and dissection of antigen-presenting cell function.

L134 ANSWER 39 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 82156828 EMBASE
DOCUMENT NUMBER: 1982156828
TITLE: Nonimmunogenic radiation-induced lymphoma: Immunity induction by a somatic cell hybrid.
AUTHOR: Yefenof E.; Goldapfel M.; Ber R.
CORPORATE SOURCE: Lautenberg Cent. Gen. Tum. Immunol., Hebrew Univ. Hadassah Med. Sch., Jerusalem 91010, Israel
SOURCE: Journal of the National Cancer Institute, (1982) 68/5 (841-849).
CODEN: JNCIAM
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 016 Cancer
025 Hematology
014 Radiology
026 Immunology, Serology and Transplantation
LANGUAGE: English
AB The cell line designated PIR-2 is a nonimmunogenic X-ray-induced thymoma of C57BL/6 origin that is unable to induce antitumor immunity in syngeneic lymphocytes in vitro and in mice in vivo. Fusion of PIR-2 with an **allogenic** 'universal fuser' A9HT (clone 3c) resulted in the establishment of a somatic cell hybrid designated A9/PIR. C57BL/6 lymphocytes sensitized in vitro with A9/PIR could lyse parental PIR-2 cells, as well as other syngeneic tumors. However, immunization of mice with the hybrid significantly enhanced PIR-2 tumor takes while it partially protected the animals against a challenge with unrelated syngeneic tumors. The results imply that somatic cell hybridization can increase the immunogenicity of an otherwise nonimmunogenic tumor. However, in view of the enhancing effects of hybrid preimmunization on parental tumor cell growth, the possible application of this approach for immunotherapy is questionable.

L134 ANSWER 40 OF 46 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 80232103 EMBASE
DOCUMENT NUMBER: 1980232103
TITLE: Construction of T cell hybridomas secreting **allogeneic** effect factor.

AUTHOR: Katz D.H.; Bechtold T.E.; Altman A.
CORPORATE SOURCE: Dept. Cell. Developm. Immunol., Scripps Clin. Res. Found., La Jolla, Calif. 92037, United States
SOURCE: Journal of Experimental Medicine, (1980) 152/4 (956-968). CODEN: JEMEAV
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English

AB T cell hybridoma lines were constructed by fusion of DBA/2 alloantigen-activated T cell blasts with the AKR thymoma line BW5147. Certain of the hybridomas prepared in this manner secreted spontaneously into their culture supernates biologically active molecules that displayed B cell- and T cell-activating properties characteristic of **allogeneic** effect factor (AEF). Cell surface phenotype analysis documented that the hybridomas were, indeed, somatic cell hybrids between the two respective partner cells used for fusion. The B cell-activating properties of these hybridoma supernates was demonstrated by their capacity to stimulate T cell-depleted spleen cells to respond in vitro to T-dependent antigens. The T cell-activating properties of these hybridoma supernates was verified by their capacity to stimulate autonomous development of self-specific cytotoxic T lymphocytes and by their capacity to exert mitogenic effects on unprimed T cells. The biologically active molecules secreted by these hybridomas were, like conventional AEF, inhibitable by specific anti-Ia antibodies thus indicating the presence of Ia determinants on the relevant hybridoma products. Finally, these AEF-secreting hybridomas could be stimulated to proliferate and to secrete increased quantities of AEF when exposed to the specific alloantigen-bearing target cells to which the T cell blasts had been originally sensitized.

L134 ANSWER 41 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2001-281836 [29] WPIDS
DOC. NO. CPI: C2001-085769
TITLE: Antigen-specific modulation of immune responses, useful for treating or preventing graft rejection, using specific regulatory T cells or their inhibitors.
DERWENT CLASS: B04 D16
INVENTOR(S): YOUNG, K; ZHANG, L; ZHANG, Z X; YANG, L
PATENT ASSIGNEE(S): (YOUN-I) YOUNG K; (ZHAN-I) ZHANG L; (ZHAN-I) ZHANG Z X; (YANG-I) YANG L
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001026679	A2	20010419	(200129)*	EN	73

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

CA 2316089 A1 20010408 (200131) EN

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001026679	A2	WO 2000-CA1172	20001006
CA 2316089	A1	CA 2000-2316089	20000824

PRIORITY APPLN. INFO: US 2000-226573 20000821; US 1999-158132 19991008

AB WO 200126679 A UPAB: 20010528

NOVELTY - Use, for suppressing an immune response, of

(i) regulatory T cells (A) having the phenotype CD3+ alpha beta TCR+CD4-CD8-CD11a+CD18+CD25+CD28+CD44-NK1.1- Ly-6A+;

(ii) an agent (I) that stimulates (A);

(iii) a Ly-6A protein (II), or nucleic acid encoding it; or

(iv) an osteopontin protein (III), or nucleic acid encoding it.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) use of an agent (IV) that inhibits (A), Ly-6A or osteopontin for enhancing an immune response;

(2) method for in vitro expansion of (A);

(3) isolated (A); and

(4) antibodies (Ab) that bind to (A).

ACTIVITY - Immunosuppressant; immunomodulatory; antidiabetic; anti-inflammatory; anti-allergic; antirejection; antimicrobial.

MECHANISM OF ACTION - Suppression or activation of a cytotoxic T cell (CTL) response in an antigen-specific manner, including induction of antigen-specific tolerance. Probably, since (A) express Fas ligand at high levels, they capture alloantigens from **antigen-presenting** cells (through the anti-host T cell receptor), turning them into killer cells. These cells, with captured antigens on their surfaces, attract activated anti-host CTL and send death signals to them through Fas ligand. The process depends on Fas/Fas ligand contact so (A) will not cause guest versus host disease themselves since most host tissues, although expressing Class I **MHC**, do not express Fas.

USE - The method is used, in human or veterinary medicine:

(a) to treat or prevent graft rejection (particularly of skin or heart); guest versus host disease; a wide range of autoimmune diseases (e.g. multiple sclerosis, rheumatism, diabetes etc.) or allergies; or

(b) when used to promote an immune response, to treat infections and acquired immune deficiency syndrome.

Antibody (Ab) that bind to (A) can be used to suppress or enhance an immune response; to isolate or purify (A), and for identifying proteins important for survival and function of (A). When B6xC.B-17 mice were injected intravenously with 30 million viable spleen cells from 2 x dm2 mice (i.e. a mismatch at only one Class I locus Ld), none of them developed guest versus host disease (GVHD) and all survived at least 150 days. When the animals were injected similarly with fully mismatched cells from B6 mice, they all developed severe GVHD and were dead within 2 weeks. Dwg.0/16

L134 ANSWER 42 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-638307 [61] WPIDS
DOC. NO. CPI: C2000-192004
TITLE: Generating antigen specific T-cells useful for treating **cancer** and viral infections comprises combining a dendritic cell and a **tumor** cell or a virally infected cell.
DERWENT CLASS: B04 D16
INVENTOR(S): FALO, L D; STORKUS, W
PATENT ASSIGNEE(S): (UYPI-N) UNIV PITTSBURGH
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

WO 2000057705 A1 20001005 (200061)* EN 29
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2000041831 A 20001016 (200106)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000057705	A1	WO 2000-US8472	20000330
AU 2000041831	A	AU 2000-41831	20000330

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000041831	A	Based on	WO 200057705

PRIORITY APPLN. INFO: US 1999-282679 19990331

AB WO 200057705 A UPAB: 20001128
NOVELTY - A method (M1) for generating antigen specific T-cells comprises combing at least one first cell with at least one second cell in vitro, where the first cell is an autologous dendritic cell and the second cell is a **tumor** cell or a virally infected cell, adding autologous T-cells to the combination, culturing the mixture and harvesting the T-cells from the mixture.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an antigen specific T-cell (I) prepared by M1;

(2) a method (M2) for effecting **immunotherapy** in a host comprising administering (I);

(3) a method (M3) of identifying antigens comprising:

(a) loading antigen presenting cells with peptides extracted from **tumor** cells;

(b) analyzing the reactivity of the antigen presenting cells with (I); and

(c) identifying the peptides recognized by (I);

(4) a method (M4) of identifying antigens comprising:

(a) transfecting cells with **tumor**-derived DNA or **tumor**-derived cDNA;

(b) screening the transfected cells of step (a) for their ability to recognize (I); and

(c) extracting transfected DNA or cDNA from the recognized cells of step (b); and

(5) generating an animal model for the study of **immunotherapy** comprising transferring one or more (I) into a **tumor** bearing host.

ACTIVITY - Cytostatic; antiviral.

No supporting biological data given.

MECHANISM OF ACTION - None given.

USE - To prepare antigen specific T-cells which can be used to treat **cancer** and viral infections.

ADVANTAGE - The T-cells prepared by the new method provide protective and therapeutic immunity to a wide variety of **tumor** types and viral **immunotherapy** towards a wide variety of viral infections.

Dwg.0/3

L134 ANSWER 43 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-105691 [09] WPIDS
DOC. NO. CPI: C2000-031722
TITLE: Obtaining antigen presenting cells from a patient for use in **immunotherapy**.
DERWENT CLASS: B04 D16
INVENTOR(S): VAN VLASSELAER, P
PATENT ASSIGNEE(S): (DEND-N) DENDREON CORP
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9963050	A2	19991209	(200009)*	EN	21
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9942269	A	19991220	(200021)		
EP 1082411	A2	20010314	(200116)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9963050	A2	WO 1999-US12142	19990601
AU 9942269	A	AU 1999-42269	19990601
EP 1082411	A2	EP 1999-926111	19990601
		WO 1999-US12142	19990601

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9942269	A	Based on	WO 9963050
EP 1082411	A2	Based on	WO 9963050

PRIORITY APPLN. INFO: US 1998-87764 19980602

AB WO 9963050 A UPAB: 20000218
NOVELTY - A method for obtaining antigen presenting cells (APCs) from a human patient who is under treatment with an agent effective to mobilize stem cells, APCs and their precursors, from bone marrow into the peripheral blood, is new.

DETAILED DESCRIPTION - The method comprises obtaining a blood cell fraction containing peripheral blood mononuclear cells, subjecting the fraction to density centrifugation, harvesting the cells at the interphase, to obtain a cell fraction enriched in precursor APC, and culturing the harvested cells under conditions effective to induce cells having the morphology, phenotype and function of dendritic cells.

INDEPENDENT CLAIMS are also included for the following:

(1) an **immunotherapy** method for treating a patient, comprising:

(a) treating the patient with an agent effective to mobilize stem cells, APCs and their precursors, from bone marrow, over a period sufficient to cause cell mobilization from bone marrow into the peripheral blood;

(b) obtaining from the patient, a blood cell fraction enriched in

peripheral blood mononuclear cells;
(c) subjecting the blood cell fraction to density centrifugation;
(d) harvesting the cells at the interphase, to obtain a cell fraction enriched in precursor APCs;
(e) culturing the harvested cells under conditions effective to induce cells having the morphology, phenotype, and function of dendritic cells; and
(f) administering the cultured, induced cells to the patient; and
(2) a human blood cell composition for use in **immunotherapy**, containing a mixture of stem cells and precursor APCs, prepared as above.
ACTIVITY - Cytostatic; antiinfectious.
MECHANISM OF ACTION - The APCs are used for **immunotherapy**.
USE - The process is used in treating **cancer** or infectious disease by **immunotherapy**, where the antigen(s) are **cancer**- or viral-specific antigen(s), respectively (all claimed).
Dwg.0/1

L134 ANSWER 44 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1999-458609 [38] WPIDS
DOC. NO. NON-CPI: N1999-343047
DOC. NO. CPI: C1999-134665
TITLE: Pure population of educated, antigen-specific immune effector cells, useful in adoptive **immunotherapy** of **cancer**, as vaccine and for isolating specific antigens.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): NICOLETTE, C A; ROBERTS, B L
PATENT ASSIGNEE(S): (GENZ) GENZYME CORP
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9937313	A1	19990729	(199938)*	EN	58
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9923392	A	19990809	(200001)		
EP 1071436	A1	20010131	(200108)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9937313	A1	WO 1999-US1464	19990125
AU 9923392	A	AU 1999-23392	19990125
EP 1071436	A1	EP 1999-903347	19990125
		WO 1999-US1464	19990125

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9923392	A	Based on	WO 9937313
EP 1071436	A1	Based on	WO 9937313

PRIORITY APPLN. INFO: US 1998-80041 19980331; US 1998-88357 19980126
AB WO 9937313 A UPAB: 19990922
NOVELTY - Pure population of educated, antigen-specific immune effector cells (A), expanded in culture at the expense of **hybrid** cells (B) that consist of **antigen-presenting** cells (APC) **fused** to cells that express one or more antigens (Ag).
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) production of (A) by culturing immune effector cells with (B);
(2) method for identifying a gene fragment (GF) that encodes an Ag recognized by (A);
(3) method for identifying a polypeptide (I) that encodes a sequence motif in an Ag recognized by (A); and
(4) vaccines containing (A) or Ag.
ACTIVITY - Antitumor.
MECHANISM OF ACTION - None given.
USE - (A) are used for adoptive **immunotherapy**, particularly of **tumors**, in vaccines and for identification/characterization of antigens (Ag). Nucleic acids encoding Ag, or its fragments, are useful in radioassays and polymerase chain reaction to detect/monitor Ag-expressing cells or tissues, e.g. in response to drugs, also in gene therapy (in vivo or ex vivo), e.g. where it encodes a dominant-inhibiting mutant of a wild-type immunosuppressant. Antibodies raised against Ag can be used to identify Ag, or its fragments, also therapeutically.
Dwg.0/2

L134 ANSWER 45 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1994-279731 [34] WPIDS
DOC. NO. CPI: C1994-127705
TITLE: Prodn. of growth enhancing media supplement for cell culture - usign paste of serum Cohn fraction IV4, contacting with buffer, adjusting pH, clarifying and filtering.
DERWENT CLASS: B04 D16
INVENTOR(S): DROHAN, W; ENOMOTO, S; MAGUIRE, Y P; MANKARIOUS, S S
PATENT ASSIGNEE(S): (AMNA-N) AMERICAN NAT RED CROSS; (BAXT) BAXTER INT INC
COUNTRY COUNT: 18
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9418310	A1	19940818	(199434)*		55
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE					
W: CA JP					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9418310	A1	WO 1994-US1522	19940214

PRIORITY APPLN. INFO: US 1993-188218 19930212
AB WO 9418310 A UPAB: 19941013
Method for producing a human-derived growth-enhancing media supplement (I) for the in vitro culture of cells comprises (a) contacting a paste comprising human serum Cohn fraction IV4 with a resuspension buffer of pH 7.4-8.4 to form a first suspension; (b) adjusting the pH to form a homogeneous suspension of stable pH 6.4-8.0; (c) clarifying to form a supernatant; and (d) filtering the supernatant through a steriliser filter

to form (I).

Also claimed is a method for culturing cells in vitro by (e) adding (I) to a basal medium to form a complete growth medium; and (f) culturing cells.

USE/ADVANTAGE - (I) is suitable for the culture of **hybridomas**, mammalian cell lines, haematopoietic cells and primary cells from normal tissue and **tumours**. (I) can replace up to 95% of the otherwise required foetal bovine serum leading to a great redn. in cost and an increase in availability and convenience. (I) can be used to generate large nos. of immune-system cells for the purposes of adoptive **immunotherapy**, whereby autologous or **allogeneic** cells are generated in culture for replacement to the patient when needed. Dwg.0/11

L134 ANSWER 46 OF 46 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1994-302209 [37] WPIDS
DOC. NO. CPI: C1994-137840
TITLE: New class 1 **MHC** restricted T-T **hybridomas** producing lymphokine - are alloreactive or antigen specific, derived from BW 5147 cells transfected with CD8 gene by **fusion** with T lymphocyte.
DERWENT CLASS: B04 D16
INVENTOR(S): ROCK, K L
PATENT ASSIGNEE(S): (DAND) DANA FARBER CANCER INST INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 5348878	A	19940920	(199437)*		23

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 5348878	A	CIP of	US 1990-521838	19900510
			US 1991-814069	19911224

PRIORITY APPLN. INFO: US 1990-521838 19900510; US 1991-814069 19911224

AB US 5348878 A UPAB: 19941109
New alloreactive, lymphokine producing, class 1 **MHC** restricted T-T **hybrids** are **fusion** products of (1) BW5147 cells transformed or transfected with a CD8 gene and able to express this gene's product; (A) and; (2) alloreactive T lymphocytes. **Hybrids** express (A) and produce lymphokines in response to antigenic stimulation with target cells bearing **allogenic** class I molecules shared by lymphocytes against which the alloreactive lymphocytes were developed, but not in response to stimulation with **allogenic** cells bearing only class H molecules. Also new are similar **hybrids** which are antigen specific rather than alloreactive. In this case (2) is an antigen-specific T lymphocyte and the **hybrid** produces lymphokines in response to antigenic stimulation with **antigen presenting** cells which share class I molecules with the host providing the lymphocytes.

These express IL-2 and contain a murine CD8 gene, partic. the Lyt 2.2 allele. Specifically claimed are ATCC HB10385 (alloreactive) and HB1086 (antigen specific; specific for OVA peptide in association will class I H-2Kb **antigen presenting** cells).

The **hybrids** are used to analyse properties of individual T cells and for studying cellular/molecular events involved in activation, e.g. identification of determinants on antigens or detecting class I molecule alterations.

Transforming BW5147 with the CD8 gene results in more efficient formation of class I restricted **hybrids**. Lymphokine prodn. in th **hybrids** can be detected simply without needing intact **antigen presenting** cells are required in standard Cr release assay.
Dwg.0/5

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